THE

WEST RIDING LUNATIC

ASYLUM

MEDICAL REPORTS.

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VOL. V.

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'Not a fact is discovered but has its bearing on the whole body of doctrine; not a mechanical improvement in the construction of an instrument but opens fresh sources of discovery. Onward and for ever onward, mightier and for ever mightier, rolls this wondrous tide of discovery.' LEWES.

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PREFACE.

The rapid and substantial advances made in recent years in our knowledge of the nervous system, in health and disease, encourage the hope that even more momentous encroachments will be effected shortly on that still vast and unknown territory that corresponds with so much of the most subtle and supreme part of man's nature. The time seems ripe for further discovery. The spirit of wise curiosity and enterprise is abroad, and that, united with scientific discernment and aided by modern ingenuity, cannot fail to conduct to important and fertile revelations. The barriers that have so long shut us out from a practical acquaintance with the action of the brain and its tributaries are being opened up, and there is no extravagance in the expectation that we shall soon be in a position to exercise effectual control over disorders that have hitherto baffled treatment.

This being so, an urgent and imperative duty is imposed upon those who have special opportunities of studying and investigating nervous diseases. It is pressingly incumbent upon them to improve their opportunities. It behoves them to add some quota to our information respecting the physiology and pathology of the nervous system. To few is it given to penetrate mysteries or to flash forth discoveries, but to many is permitted to prepare the way for such enlightenment. Every fact faithfully observed, every experiment judiciously performed, is a step gained on the path of progress.

It is upon Asylum medical officers that the obligation to watch and interrogate nervous diseases most heavily falls, for their opportunities of doing so are peculiarly great and excellent. Our lunatic hospitals are stored with only too vast an accumulation of pathological material, while their organization affords unusual facilities for observation and research. The public are entitled to look to them and their medical officers for no small subsidies to scientific medicine, and for practical aid in stemming the great and growing tide of insanity and allied diseases that overwhelms so many valuable lives, that does so much damage, and creates such onerous burdens. That the medical officers of the West Riding Asylum are alive to their obligations in these respects, and are anxious to meet them, the present volume will, perhaps, be accepted as a proof. It contains the results of some of the inquiries which they have undertaken in that institution during the past year, as well as contributions by other distinguished workers in the field of nervous pathology who are not officially connected with the West Riding Asylum, but who sympathize with the efforts which are being made in it to advance our knowledge of cerebral and nervous derangements and degenerations.

INFLUENCE OF DIET IN EPILEPSY.

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THE primary object of this Essay is to record the results of a series of observations on a number of patients suffering from epilepsy, undertaken with the view of determining the relative value of a nitrogenous and a farinaceous diet in the treatment of that disease. These observations were suggested by a paper in the third volume of the West Riding Asylum Medical Reports on the 'Investigation of Epilepsies,' by Dr. Hughlings Jackson, in which the author brings forward certain speculations regarding the nature of the morbid alteration in the nutrition of brain tissue, underlying the epileptic condition. These speculations I shall subsequently refer to at more length; in the meantime it will be sufficient to state that they lead Dr. Hughlings Jackson to the conclusion that a farinaceous diet is more suitable in the epileptic condition than a nitrogenous. It was for the purpose of testing the value of this opinion that I undertook the series of investigations which it is the object of this paper to record.

Before giving the results obtained, it may not be out of place to discuss briefly the relation of food to the forces displayed in the living body, and to enquire how far and in what way the nutrition of the brain may be influenced by the quantity and quality of the food.

The physiology of the food, in its relation to the forces manifested in the living body, is a subject that has of late

years assumed an importance and significance that it did not previously possess. The progress of scientific enquiry in this direction is due in a great measure to the discovery of the law of the correlation of forces, which was first enunciated in this country by Grove in his work on the ' Correlation of the Physical Forces.' In that Essay Grove demonstrated that one kind of force was capable of producing another, and sought to establish the position that the physical forces, heat, light, electricity, magnetism, chemical attraction, and motion, are all reciprocally dependent, in other words, that either of these forces might produce the other, or in its turn be produced by any of the others. When expressed in its complete form, this principle, which is now more accurately designated the law of the ' Conservation of Energy,' sets forth that the whole amount of energy in the universe, like that of matter, is a fixed quantity ; that, like matter, energy is indestructible and cannot undergo either increase or diminution, however variously it may be modified or transformed. The law further implies that each kind of energy has its definite equivalent in other forms, so that no force can be made to disappear in one shape without an equivalent amount in some other form coming into existence. So much electrical energy, for instance, will always, under the same circumstances, be generated by the same amount of mechanical power, which again will be represented by a definite quantity of heat, and so on.

The principle of the ' Conservation of Energy' being accepted relative to the purely physical forces, will be found equally applicable when extended to the realm of organic nature. At first sight the phenomena of life and growth in the plant or animal present something very like an origination of force *de novo* ; but when the matter is investigated in the light of modern scientific discoveries, it will be found that we have here only another instance of transformation of energy from one form to another. Vegetable nature is dependent for its sustenance on principles derived from the inorganic world, chiefly in the form of carbonic acid and ammonia ; but the plant has not the power in itself of appropriating these substances in the form in which they are

presented to it. Before they can become subservient to the nutrition and growth of the plant, the elements entering into the composition of these principles must be separated—in other words, chemical affinity has to be overcome. In the case of carbonic acid, for instance, only the carbon is required by the plant, and before it can be assimilated it must be disengaged from the oxygen with which it is combined in the carbonic acid. To effect this result the operation of some force is required. Now the force so employed has its origin in the heat and light emanating from the sun's rays. It has been shown that when the sun's rays are excluded, this disengagement of the oxygen from the carbon does not go on. The effect cannot, therefore, be due to the forces peculiar to the living plant. These forces may be said to have merely a directive influence, guiding, so to speak, the energy of the sun's rays to expend itself in the separation of the elements which exist united in the carbonic acid. Thus we see that in the growth of plants solar energy is employed to disengage the elements required from their natural affinities, placing them in a position of potential energy with regard to each other; in other words, the solar energy becomes transformed into potential chemical energy. The amount of solar energy so employed may be said to become fixed and stored up in the resulting vegetable product, ready to be liberated again on exposure to favourable conditions, and when these conditions occur, the amount of energy liberated will be neither more nor less than the equivalent of that used in the formation of the original product. Thus when a piece of wood or coal is burned, the heat liberated is simply the equivalent of the solar energy employed years or ages ago in unlocking the elements in the inorganic combinations from which these materials were produced.

The products of vegetable life, then, may be regarded as a vast storehouse of accumulated solar power, and upon the principle of the conservation of energy, that force which has been employed in overcoming the chemical affinity of the elements in the inorganic combinations from which plants are built up, must exist in a latent or potential state in the resulting compounds. In accordance with the same prin-

ciple, an equivalent amount of energy must re-appear, in one shape or other, whenever these compounds are subjected to conditions favourable to their return to the inorganic state, which may be regarded as the condition of stable equilibrium of their elements. To express the same thing in another form, the solar energy employed in developing combustible compounds by the disengagement of oxygen from oxidised principles, re-appears in an active state and in equivalent amount when combustion of these compounds takes place.

This position once established, the extension of the law of the conservation of energy to the forces manifested in animal life, is not a difficult step. We have seen that in the products of vegetable life we have a store of potential energy derived from the sun's rays during the development and growth of the plant. Now these products, either directly or indirectly constitute the food of animals, and thus a store of accumulated force is introduced into the living body. Let us see what becomes of this force. Oxidation, as we have seen, in ordinary circumstances, sets free an amount of force equivalent to that used up in the formation of the original product, and if this oxidation occurs within the animal body the result must be the same as far as the amount of force liberated is concerned. Now, if we compare the substances discharged from the system with the food ingested, we find that such an oxidation has taken place. The excretions are the metamorphosed products of the matter taken into the system, and, supposing the weight of the body to remain constant, the elements excreted must be exactly the same in nature and quantity as those taken in, since there is no such thing as creation or annihilation of matter. There is however a vast difference in the forms of combination of the elements in the two cases. In the food we have compounds of highly complex chemical composition, unstable in their constitution and having a tendency under favourable circumstances to undergo oxidation and liberate an amount of force. In the excreta, on the other hand, the elements form comparatively simple combinations of a more stable character, and, in the case of some of the products at least, incapable of undergoing further oxidation. This change has been

effected by the operation of the living body. The natural affinities of the elements composing the food have become to some extent satisfied, and the force previously employed in overcoming those affinities, must, in accordance with the law of the conservation of energy, have re-appeared under some form of manifestation or other. For the equivalent of the force thus liberated we must look to the various manifestations of energy exhibited in the actions of animal life, such as muscular contraction, animal heat, nutritive and assimilative action, and nervous energy. These actions, therefore, do not represent a creation of force under the influence of vital activity, as might at first sight be supposed, but owe their existence to a transformation of the potential energy stored up in the food introduced into the system ; and if we had any measurable standard by which to compare those forces manifested in the living body, we should find that, in the long run, the sum of their equivalents would be the exact representative of the force liberated from the food by the descent of its complex principles from the condition in which they are ingested to the more simple combinations found in the excretions.

So far we have spoken of food only in its relation to the liberation of force by the process of disintegration to which it is subjected. But all the food does not *directly* undergo this process. Some of it has a higher destination. The transformation of the energy of the food into those forces peculiar to the animal system, can take place only through the medium of specialised structures made up, in some cases, of highly complex principles. These tissues, in the production of their particular modes of energy, are subjected to waste and disintegration, and this waste has to be repaired by the assimilation of the appropriate material from the food. The material required for this purpose is in some cases contained in the food in the suitable form, but in others has to be elaborated from more simple compounds, and this elaboration is effected by the application of the force liberated from that portion of the food which has undergone retrograde metamorphosis. Thus we see that, while one portion of the food becomes at once disintegrated, liberating an amount of

force which is applied in various directions, another portion experiences a progressive metamorphosis, resulting in the development of highly specialised structures, which by undergoing disintegration in their turn, become the means of evolving special and peculiar modes of activity.

This specialisation of structure and function reaches its highest development in the brain and nervous system, where we have to deal with tissues made up of a large number of heterogeneous principles of great complexity, manifesting a special and peculiar mode of energy, with the exact nature and relations of which we are still unacquainted. We are far from being able to trace even the sequence of events by which the principles of the food are raised to this highest plane of organisation, but we may rest assured that, here as elsewhere, the principle of the 'Conservation of Energy' holds good, and that, if we could trace the food through all the changes and transformations which it undergoes, from the time when it enters the body till it becomes a constituent part of the brain, we should have a series of transmutations characterised by the same simplicity and the same economy of force as we see displayed in other operations of nature which are more amenable to our present methods of observation. Thus, though it would seem at present a bold step to refer the vital manifestations of voluntary movement and sensation to the same category as physical and chemical phenomena, and to trace back the operations of the mind which can recognise the wonder of its own being, to a mere transmutation of the energy derived from the food, there can be no doubt that to such energy are due the existence and maintenance of the special arrangement of matter by which alone nervous and psychical activity can be manifested, and that every manifestation of nervous activity has its corresponding physical expression in the molecular or chemical changes of the material substratum.

From what has been said, it will be apparent that the healthy activity of nervous tissue may, in a great measure, be determined by the nature of the nutriment introduced into the system. There is little doubt that the mode of activity peculiar to the nervous system is in some way de-

pendent on or related to the force stored up in the food. The exact nature of that relationship, however, and of the processes by which nervous energy is evolved out of other modes of energy, must, in the present state of our knowledge, remain a sealed book.

If, as seems probable, nerve force is the result of the interplay of chemical and molecular changes, it appears to me that the only hope of our being able to solve the question of its relation to other forces lies in a thorough knowledge of the molecular constitution and chemical relations of those tissues whose special function it is to store up and expend nervous energy. It is through this channel, too, that we can alone obtain anything like a rational explanation of the influence of therapeutic and dietetic agents on the functions of the nervous system. Our knowledge of the chemistry of the nervous system has hitherto been vague and uncertain. The latest investigations on the subject, however, promise to be attended with more satisfactory results. I refer to the elaborate researches of Dr. Thudichum, an account of which has been lately published in the Reports of the Medical Officers of the Privy Council for 1874. Dr. Thudichum's analyses are not merely interesting to the chemist, but bid fair to be attended with practical results of the highest importance, both to physiology and therapeutics. He arranges the immediate principles found in the brain in five groups, according to the complexity of their composition. The first group consists of *principles containing five elements*, C.H.N.S.O., and is termed the group of *sulphurised or albuminous principles*. The second group also consists of *principles containing five elements*, C.H.N.P.O. This he terms the *group of phosphorised principles*. It seems to correspond to the protagon of Liebreich and Bayer, and includes three well-marked sub-groups, *kephaline, myeline*, and *lecithine*, the chemical characters of which Dr. Thudichum summarises as follows:—'The kephalines possess the tendency to be oxidised—oxidisability; the myelines are not easily changed by any agency or influence, and therefore possess stability; the lecithines easily fall to pieces; they are afflicted with lability.' In the third group are included a number of *prin-*

ciples containing four elements, C.H.N.O., the nitrogenised principles. They include cerebrine, kerosine, phrenosine, and a variety of other bodies. The fourth group consists of *principles containing three elements*, C.H.O., and is termed the oxygenated group. It includes the varieties of cholesterine, hydrocarbons, such as inosite, organic acids, fats, and fatty acids. The fifth and last group is that of *inorganic principles*, including acids, bases, and salts, both free and in combination with many of the immediate principles belonging to the other groups.

Of the part which each of these groups of principles plays in the evolution of the mode of activity peculiar to the brain we know almost nothing; but the information already obtained by Dr. Thudichum regarding the chemical relations of some of them justifies us in expecting results of the highest value in future researches. The albuminous compounds form an essential constituent in the composition of the brain as of other tissues, and no doubt play an equally important part in the evolution of its special activity as they do in the case of the other living tissues. Throughout the whole realm of organised nature, wherever we find active vital changes going on, albuminous principles are present in one form or other, and constitute the principal medium through which vital phenomena are manifested. So it is in the brain. Albuminous matter is said to constitute about eight per cent. of the entire mass of the brain, that is, about one-third of the solid constituents. Of the behaviour of these substances while in combination in the fully constructed brain we can, of course, know nothing except by inference from its behaviour when isolated. Now it appears that brain albumen when isolated presents no specific differences from the albuminous matters of the body in general; but Thudichum states that when placed in the presence of some of the peculiar brain matters, the behaviour of soluble albumen is different from that which it ordinarily observes; and that the change thus induced is due to an influence of these brain matters amounting almost to chemical combination. It would, therefore, appear probable that in the brain the behaviour of the albuminous compound is, to some ex-

tent, determined and governed by the other principles peculiar to the brain. The chief of these are to be found in the group of phosphorised bodies, which are evidently of great significance in relation to the evolution of nerve force. Thudichum has shown that some of these substances when isolated manifest a diversity of chemical affinity, such as is possessed by no other substance in nature at present known. He found, for instance, that they possessed alkaline, acid, and alkaloid affinities; that all these affinities were diminished in the presence of water, and some of them overcome by water in quantity. Their affinity for water again may be overcome by the metallic oxides of lead, copper, manganese, iron, and to a smaller extent by those of lime and potash. These latter compounds can be dissociated by the strong mineral acids, and the compounds again separated by dialysis. The nitrogenised group of principles are stated to exhibit in many respects, though to a less degree, the same properties as the phosphorised.

With such a variety of chemical affinities, it follows that the phosphorised principles must respond to the slightest external chemical influence which may reach them through the circulation. A change in their chemical relations is thus induced which reacts upon the other brain substances, inducing in them changes multiform in proportion to the varied affinities of the substance originally affected. In the diversity of changes thus brought about we see a faint representation, from the chemical side, of the wonderful manifestations of activity which are exhibited in the vital functions of the nervous system.

Considering the manifold affinities possessed by nerve matter, and its consequent liability to be affected by whatever influence may reach it, the question of the supply of food in relation to the nutrition of the brain becomes a most important one. We have seen that the purpose fulfilled by food in the system is two-fold, first, to supply material for the formation of living tissues, and secondly, to evolve force which is applied to raising the former portion to the condition of living matter, and to other vital processes. There would seem, therefore, to be two ways in which we can affect the

nutrition of any particular organ through the food, first by increasing or diminishing the amount of principles from which the tissues of the organ in question are built up; and secondly, by so regulating the quantity and quality of the food as to affect the general nutritive activity of the system. In the case of the brain the constitution and chemical relations of its constituent principles are so complex that it is difficult to trace their connection with the food, or even to say from what principles in the food some of them are derived. We have seen, however, that compounds containing nitrogen in one form or other make up a large proportion of the entire brain substance, a fact that leads us naturally to expect that the nutrition of this organ may be most readily affected through the increase or diminution in the nitrogenous principles of the food. The phosphorised matters of the brain are also marked out by their multiform chemical relations as likely to play an important part in the production of nervous activity, and in this relation the amount of phosphorus in the food becomes an important consideration. In the present state of our knowledge it is not very obvious from what constituent of the food phosphorus is assimilated by the brain. In all probability, however, it is derived chiefly from that contained in combination in the nitrogenous compounds; and if so, we have a still further reason for regarding the nitrogenous material of the food as of most importance in reference to the healthy nutrition of nerve tissue. It is impossible to say what part the non-nitrogenous elements of the food play in the production of nerve tissue and the evolution of its activity. It may be that some part of it is turned to account in the formation of the brain substance, and in the process of nutrition peculiar to these tissues. It may be also, that by the peculiar action of living nerve tissue, the forces of the non-nitrogenous materials are transformed into nerve force, just as we know that they are turned to account by the muscles in the production of mechanical power. On this subject, however, we have at present no information, and we must leave the solution of the question to future researches.

Another way in which the nutrition of the brain may be

influenced through the food is by the effect of the latter on the circulation and the general nutritive activity of the system. Dr. Parkes has proved that the power of the heart's action is capable of being diminished by a reduction of the nitrogenous principles of the food, especially if the voluntary muscles are kept in active exercise. A man was fed for five days on a diet of non-nitrogenous matter, consisting of fat and starch alone. It was found that the voluntary muscles, excited by the will, could act with as much force as ever, although starved of nitrogenous matter; but it was not so with the involuntary muscles. The heart soon began to suffer in nutrition, and its force, as shown by the sphygmograph, was reduced nearly one half. 'I draw the conclusion from this experiment,' says Dr. Parkes, 'that if the nitrogen is cut off, and the voluntary muscles are kept in their usual action, they do not fail, but that the power of the heart may be thus reduced if it be desired to do so. In other words, the food the heart requires is attracted from it by more potent actions. Here, then, we possess a power of affecting the action of the heart if it be needed.' The same remarks will, of course, apply to the muscular tissue throughout the circulatory system. There can be little doubt, then, that the condition of the circulation in the brain, and through that the activity of its nutrition, may be influenced by the amount of the nitrogenous elements of the food.

In considering the question of diet in its bearing on the treatment of epilepsy, we have first to enquire into the probable nature of the abnormal condition of brain matter existing in that disease, and then to determine whether this condition is likely to be influenced by the quantity and quality of the food in either of the ways previously mentioned. The enquiry is beset with difficulties, and cannot be prosecuted with satisfactory results until we know more of nerve matter and its peculiar mode of energy than we do at present. Indeed it may safely be said that our present knowledge of the nervous system warrants only conjectures as to the real nature of the morbid alteration in nutrition existing in the epileptic condition. We know, however, that in the epileptic brain the grey matter is so abnormally

nourished that it occasionally reaches very high tension, so to speak, and discharges in an irregular and spasmodic manner. We must suppose that there is a tendency to the development of more nervous energy than in health, in other words, there is an abnormal increase in the nutritive activity of the brain. Now, we have seen that there are good grounds for the belief that the mode of energy peculiar to the brain has its source chiefly in the albuminous and other nitrogenised principles. If then there exists an abnormal tendency to increased evolution of such energy, there will be a greater demand on the nitrogenous elements of the food, and it is easy to conceive that an excess in the quantity of these elements would keep up and increase the abnormal tendency already existing. On the other hand, the effect of limiting the amount of nitrogen in the food would be to decrease the tendency to abnormal activity of nutrition, and thereby render the nerve substance more stable. The influence exerted on the circulation would tell in the same direction.

According to Dr. Hughlings Jackson, there is in the epileptic condition not merely an abnormal increase in the nutritive changes of the nervous centres, but nutrition is carried on on a lower level.[1]   'There are,' he says, ' two ways in which nutrition may be imperfect—in quantity and in quality. I believe that nerve tissue in discharging lesions is over-nourished in the former sense, and worse nourished in the latter. . . . I believe that the highly unstable nervous matter of disease (the discharging lesion) differs in composition, but not in constitution, from the comparatively stable grey matter of health. The alteration in composition is, of course, such that the nervous substance formed is more explosive. We must suppose that there is some order in this substitution-nutrition, and we must infer that it is in the direction of explosiveness or instability. The following is a speculation as to the kind of alteration of composition. One striking constituent of nervous matter is phosphorus. It belongs to the chemical class of triads, of

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[1] ' West Riding Asylum Reports,' vol. iii. p. 326.

which other members are nitrogen and arsenic. My speculation is that in the abnormal nutritive process producing unstable nervous matter, the phosphorus ingredient is replaced by its chemical congener nitrogen. There is a substitution compound ; the replacement probably occurs in different degrees, as it does in the three differing chloracetic acids. If nitrogen be substituted, as supposed, we can easily understand that the substance produced would be more explosive. . . . The nutrition is, therefore, assumed to be defective not in quantity but in quality, in those functional alterations I call discharging lesions.'

In accordance with these speculations, Dr. Hughlings Jackson advises that the diet of epileptics should contain only a limited amount of nitrogenous principles ; and it was with the purpose of testing the practical value of this advice that I commenced the series of observations, the results of which, as far as they have gone, I shall now proceed to record.

Twenty-four chronic epileptic patients were selected and subjected to observation under two different kinds of diet, the one consisting largely of nitrogenous matter, and the other containing no animal food, but consisting of milk, arrowroot, potatoes, butter, oatmeal, and bread. Though the terms are not strictly accurate, I shall distinguish the former as *nitrogenous* and the latter as *farinaceous*. The exact dietary adopted in each case was as follows :—Nitrogenous—*Breakfast*: 8 oz. lean meat, beef or mutton, 3 oz. bread, one pint coffee. *Dinner*: 12 oz. lean meat, beef or mutton, 7 oz. bread, varied occasionally with fresh vegetables. *Supper*: 4 oz. bread, 4 oz. cheese, and tea. Farinaceous— *Breakfast*: 8 oz. bread, ¾ oz. butter, one pint coffee. *Dinner*: On two days of the week, 1½ pint arrowroot milk, containing 2 oz. arrowroot and 3 oz. bread ; on two days, oatmeal porridge, containing 3 oz. oatmeal to each man, with a pint of milk ; on the remaining three days 12 oz. potatoes, 1½ oz. butter, 3 oz. bread, and half pint milk. *Supper*: Same as breakfast, except that tea was substituted for coffee.

The following tables represent the average amount of the various nutritive principles in the dry state contained in the

quantity allotted to each man per day. Though not pretending to anything like absolute precision, they will give an approximate idea of the value of the two kinds of diet, and serve as a basis of comparison between the two. The amount of the solid principles in each article of food has been calculated in accordance with the tables given in Dr. Pavy's work on ' Food and Dietetics,' the result being expressed in grains.

Nitrogenous Diet.

				Nitrogenous Matter	Fats	Carbohydrates	Salts	Total
Breakfast	.	.	.	783	115	780	166	1844
Dinner	.	.	.	1667	229	1560	332·4	3788·9
Supper	.	.	.	585·7	333	891·5	89	1899·2
Total	.	.	.	3035·7	677	3231·5	587·4	7532·1

Dynamic value 2,981 foot tons.

Farinaceous Diet.

				Nitrogenous Matter	Fats	Carbohydrates	Salts	Total
Breakfast	.	.	.	245	342	1260	70·4	1917·4
Dinner	.	.	.	629·6	694	2170	131·8	3625·4
Supper	.	.	.	245	342	1260	70·4	1917·4
Total	.	.	.	1119·6	1378	4690	272·6	7460·2

Dynamic value 3,151 foot tons.

A comparison of the above tables will show that the weight of dry nutritive principles in the food consumed in the two cases was pretty nearly equal, being 7,460·2 grains per diem in the one case and 7,532 grains in the other. Leaving the salts out of consideration, the nitrogenous matter represents nearly a half of the entire amount in the first table, whereas in the second the nitrogenous principles form scarcely a fifth part. The dynamic value, calculating all the nitrogenous material as albumen, was found to be 2,981 foot tons in the nitrogenous diet, and 3,151 foot tons in the farinaceous, in other words the force produced by oxidation within the body was such as, when converted into its equiva-

lent of mechanical power, would suffice to lift 2,981 tons in the one case and 3,151 tons in the other through a distance of one foot. The excess in the dynamic value of the farinaceous over the nitrogenous is due to the limited amount of fatty material in the latter, and to the excess in the quantity of salts, which are counted as neutral.

It will be observed that in both cases the quantity is considerably less than what is usually considered as the average diet necessary for a healthy adult with moderate exercise. In this case, however, the amount of exercise was limited. The patients under observation were engaged in light occupation in the wards, and walking exercise in the airing grounds when they were inclined. The amount of actual mechanical energy expended was therefore very small.

Previous to commencing the above dietary each patient was accurately weighed, and his physical and mental condition noted. Twelve of the patients under observation were then put on nitrogenous diet, the other twelve on farinaceous, and this arrangement was continued for four weeks, during which time their physical and mental conditions were closely observed from day to day, and the number of epileptic seizures accurately noted. At the end of four weeks the weights were again taken and the diet changed, those previously on nitrogenous diet being transferred to farinaceous, and those on farinaceous to nitrogenous. After another period of four weeks, during which the condition of the patients was from time to time noted and compared with that which had been observed in the previous period, the weights were again ascertained, and the patients allowed to resume their ordinary dietary.

The results obtained during those two months of observation are not such as to justify the conclusion that either of the two diets used possessed any decided advantage over the other in the treatment of epilepsy. A reference to Tables I. and II. (pages 20 and 21), however, will show that as regards the number of fits in individual cases, there is a slight balance in favour of the farinaceous regimen. I shall afterwards analyse at greater length the results there tabulated, but before doing so, I wish to refer shortly to certain ob-

servations made on the general mental and physical condi-
tion of the patients under treatment. And first, as to the
mental condition.

In a considerable number of those who took nitrogenous
food during the first month, it was observed that soon after
commencing that diet they became much more dull and
stupid than they had previously been, would sit in a dreamy,
listless manner for a great part of the day, were very slow
and languid in their movements, and took little notice of
what was going on around them. This condition was espe-
cially marked in the first four Cases in Table I., and was pre-
sent in a slighter degree in the Cases numbered 8, 10, and 11.
It remained more or less throughout the whole period of
nitrogenous food, though it varied in intensity at different
times. No relation was traced between it and the recur-
rence of fits. As soon as the diet was changed to the farina-
ceous, it was remarked that the condition of mental hebe-
tude began to pass off, and in some of the cases the change
was very remarkable. The improvement in the mental con-
dition was not always accompanied by any marked diminu-
tion in the number of the fits. In Case No. 3, for instance,
where the mental improvement was perhaps more decided
than in any other, the number of fits during the month of
farinaceous diet was only two less than that during the
month of nitrogenous food, being 48 in the latter and 46 in the
former. A similar condition was observed in some of those
who began with farinaceous food. Several patients who,
during the period of farinaceous diet, had been observed as
more lively and intelligent, became languid and stupid when
the food was changed to the nitrogenous. This was the case
more particularly in Cases 2, 3, 5, 8, and 9 in Table II. In
some of these it will be seen that the number of fits also
was considerably increased under the nitrogenous diet; in
others, however, it was diminished, so that no connection
could be traced between this condition of mind and the
number of fits; indeed, in the case in which this stupor was
most marked, Case 8, the number of epileptic seizures was
less than during the farinaceous period. It would seem not
improbable that in some cases this condition of mental in-

ertia may have taken the place of the more active discharge of an ordinary epileptic seizure. We can quite conceive that in cases where instability of nervous matter exists and has been of long standing, a condition of the higher nervous centres may sometimes be produced in which energy is not developed of the kind necessary to call into play the special functions of these higher centres; in other words, the brain matter is so unstable that, before attaining to the degree of organisation necessary to the production of the highest form of nervous activity, it undergoes molecular change, and by descending, so to speak, to a lower plane of organisation liberates the energy stored up in it in some lower form. To this cause may possibly be referred the mental dulness and slow, languid movements of chronic epileptics. If, however, the force thus liberated does not result in mental work or in movement, it must, in accordance with the law of the conservation of energy, show itself in some other form. The form it assumes is probably that of heat, and, if so, we should look in these cases for a rise in the temperature of the body. In the cases under treatment on this occasion no observations on the temperature were taken with special reference to this point; but it will be seen that in general a higher temperature was found under the nitrogenous than under the farinaceous regimen. In another case that I have since observed, in which a condition of stupor seemed to take the place of a fit or series of fits, I noticed a rise in temperature which could not be accounted for in any other way.

In this connection the great rise in temperature observed in the *status epilepticus* is significant. In a case lately under my observation, in which the patient died after a rapid succession of severe epileptic seizures, this rise was very remarkable. During the period of the occurrence of fits the temperature varied from 104° to 105·5°. After all convulsions had ceased, it fell gradually for three hours to 103°, which was the lowest point registered. It then began to rise rapidly, and continued rising till the time of the patient's death, which took place about seven hours after the cessation of the fits. An hour before death the temperature in the axilla was 107·8°. There was here then, it will be observed,

a decided fall after the cessation of convulsive movement, and this was followed by a rapid rise without any return of convulsions. My speculation is that this rise was due in some measure to a discharge of a low form of nerve force, which was going on all the time, though not manifesting itself in muscular movement. A similar rise of temperature may be observed in cases of so called suppressed epilepsy. I think, then, that in all these cases the development and discharge of nerve force is not suppressed, as it seems to be, but takes place on a lower level, and that the high temperature manifested is due in some measure to a transformation of this low form of nervous energy into animal heat.

The general physical condition does not appear to have been appreciably affected by one diet more than the other. Under both diets there was a slight tendency to lose weight during the first month, and this is in accordance with what has often been observed, that under a regulated diet of any kind there is a tendency to lose weight. During the second month most of the cases remained stationary in weight or gained slightly. In no case, however, was there any great variation one way or the other.

The temperature presented great variation in individual cases, some cases having a higher temperature under nitrogenous food than under farinaceous, some giving an opposite result. The evening temperature was uniformly higher than the morning, and the figures recorded were on the whole somewhat higher than normal. Taking the average of the whole twenty-four cases, the morning temperature was 0·2° higher under nitrogenous food than under farinaceous, being 98·7° in the former and 98·5° in the latter. The average evening temperature presented a similar difference, being 98·7° under farinaceous food and 98·9° under nitrogenous. This result is not what was to be expected considering the composition of the diet in each case. The dynamic value of the farinaceous was somewhat higher than that of the nitrogenous—a fact which, *cæteris paribus*, should give a higher temperature in a period of farinaceous diet. This may have some connection with the condition of stupor previously noticed as having occurred during the nitrogenous period.

The urinary secretion was examined in twelve of the cases, and it may be interesting to give a brief summary of the results obtained. It was found a difficult matter to collect all the urine in many cases, so that absolute accuracy cannot be claimed for the figures given. This, however, will not affect the comparative result, which is the chief thing of interest at present. The entire amount, as far as possible, was collected for three successive days in each case, and an analysis made of the mixed secretion. The average quantity collected in the period under nitrogenous diet was 2,960 centimetres, and under farinaceous diet somewhat higher, namely 3,050 centimetres. The amount of urea was of course much higher under the nitrogenous regimen than under the farinaceous, being in the former case 65·4 grammes for the period of three days, and in the latter 43·12 grammes. The salts, with the exception of the chlorides, were all considerably increased under the nitrogenous fare. The phosphates estimated as phosphoric acid ($P_2 O_5$) gave an average of 6·72 grammes under farinaceous food, and 7·43 grammes under nitrogenous. Similarly the sulphates, estimated as So_3, gave 4·74 grammes under farinaceous diet and 5·72 under nitrogenous. The chlorides, estimated as Na Cl., on the other hand were slightly higher during the farinaceous period than during the nitrogenous. In accordance with the increased amount of urea and salts, the specific gravity was much higher in the urine collected during the period of nitrogenous food.

We come now to consider the influence of the two different kinds of diet on the actual number of fits. It must be allowed, however, that the time over which these observations extended was too short to warrant any decided conclusion as to the advantage of the one diet over the other. In any case the number of fits can only give a rough idea of improvement or deterioration in the condition of the patient; but if we find an improvement as regards the number of fits coinciding with improvement in mental and general health under any form of treatment, there is fair reason to regard that treatment as exercising a beneficial

influence. We have seen that in many cases there was a very decided mental improvement under the farinaceous dietary, and that the general health was as good as under nitrogenous fare. We have now to notice the number of fits occurring under each kind of food.

In the accompanying tables I have drawn up in a tabular form the number of fits that occurred in each week of the period during which the cases were under observation. The first table includes those cases in which the nitrogenous food was given during the first month, the second those who commenced with the farinaceous food.

An examination of Table I. will show that out of the twelve cases, nine show a decrease in the number of fits occurring during the four weeks of farinaceous food, as compared with the number during a similar period of nitrogenous diet. In some of these the decrease is very considerable. The average number of fits in each of these nine cases is 26·2 for the period of nitrogenous food and 19·8 for that of farinaceous. Of the remaining three cases, one had the same number of fits in each period, another had one more under farinaceous regimen than under nitrogenous. In the third case the number under farinaceous diet was more than doubled. This patient is, however, subject to periodic outbursts of fits, and the case cannot, therefore, be fairly regarded as telling against the farinaceous diet.

TABLE I.

No	Initials	Number of Fits on Nitrogenous diet					Number of Fits on Farinaceous diet				
		1st Week	2nd Week	3rd Week	4th Week	Total	1st Week	2nd Week	3rd Week	4th Week	Total
1	J.M.	11	2	1	1	15	3	1	2	0	6
2	F.R.	2	4	6	3	15	2	2	1	1	6
3	H.K.	16	12	10	10	48	15	9	10	12	46
4	I.K.	6	7	4	7	24	7	5	2	3	17
5	J.N.	1	6	1	1	9	3	6	0	1	10
6	E.B.	1	2	3	6	12	2	2	4	2	10
7	I.S.	0	2	1	0	3	0	2	1	0	3
8	B W.	0	7	2	5	14	3	3	1	4	11
9	I.P.	2	3	1	3	9	0	1	2	1	4
10	B.B.	6	12	23	41	82	13	26	23	7	69
11	G.L.	1	2	6	8	17	1	1	7	1	10
12	H.B.	23	11	17	12	63	15	10	69	59	153

TABLE II.

		Number of Fits on Farinaceous diet					Number of Fits on Nitrogenous diet				
No	Initials	1st Week	2nd Week	3rd Week	4th Week	Total	1st Week	2nd Week	3rd Week	4th Week	Total
1	B.L.	1	1	1	1	4	0	6	1	1	8
2	I.W.	6	2	1	9	18	1	2	8	1	12
3	J.T.	0	0	13	19	32	25	9	3	3	40
4	T.H.	0	2	2	0	4	2	0	2	0	4
5	W.G.	16	1	7	8	32	13	17	3	10	43
6	P.G.	6	4	3	1	14	2	4	2	6	14
7	W.G.	1	7	0	5	13	9	2	1	1	13
8	G.S.	8	1	3	3	15	1	3	4	4	12
9	S.F.	10	11	10	5	36	19	8	5	15	47
10	I.H.	4	5	1	3	13	1	3	1	4	9
11	I.T.M.	1	0	1	2	4	6	3	2	3	14
12	J.L.	60	70	64	65	259	55	18	26	5	116

The cases in Table II. show less decidedly in favour of farinaceous diet, but on the whole there is a slight tendency in the same direction. Out of twelve cases there was a decrease in the number of fits in five. The average number of seizures for each patient was 30·4 during the nitrogenous period, and 21·6 during the farinaceous. Of the remaining seven, three had the same number of fits during both periods, and in four there was a smaller number of fits during the nitrogenous period. In one of these the difference was very marked, the number being 259 in the farinaceous period and 116 in the nitrogenous. This patient, however, is subject to waves of severity as regards the number of fits. It is also worthy of remark that his fits during the farinaceous period were very slight; and though he was taking from eight to twelve seizures daily, his mental condition was clearer than it had been in previous attacks of a similar nature.

It will thus be seen that out of twenty-four cases observed there was in fourteen a decided decrease in the number of fits during the period of farinaceous diet; the average number of seizures for the farinaceous period in these fifteen was 20·7, as compared with 28·3 for the nitrogenous period. Of the remaining ten cases, four had the same number of fits under each diet. There are, therefore, only six left in which the advantage is on the side of the nitrogenous regimen, and

of these we have seen that two cannot be fairly regarded as telling against farinaceous diet. I think then that, after making due allowance for the short period of observation and the limited number of cases observed, there is still a certain indication that the actual number of fits is less under a farinaceous dietary than under a nitrogenous.

In a previous paragraph I stated the views entertained by Dr. Hughlings Jackson regarding the condition of abnormal nutrition existing in the substance of the epileptic brain. He believes that nutrition is carried on on a lower level, and he speculates that this lower level may consist in the assimilation of nitrogen instead of phosphorus. If this view is correct, we should expect that, if nitrogen is withheld by the restriction of the diet to farinaceous materials, and at the same time phosphorus supplied in a form available for brain nutrition, the results would be more satisfactory than those obtained with farinaceous diet alone. With the view of determining this point a further experiment was made in the case of twenty-two of the patients previously under treatment. They were put upon the same farinaceous diet as that previously employed, phosphorus being at the same time administered internally. The form of administration used was the phosphorated oil of the British Pharmacopœia made into an emulsion with mucilage and peppermint water. The dose given was ten drops of the phosphorated oil three times daily.

A month's observation under this treatment gave results of the most opposite character in different cases. In the majority of cases, however, the result was less favourable than that obtained under a farinaceous diet without phosphorus. In three cases violent explosions of fits occurred, and in nine others the number of fits was greater than that observed in the same cases under simple farinaceous food. In the remaining ten cases there was, as regards the number of fits, a balance in favour of phosphorus treatment, in most cases slight, but in one or two very considerable. As compared with nitrogenous food, the farinaceous diet with phosphorus had the advantage in fourteen of the twenty-two

cases treated, or about the same proportion as told in favour of the simple farinaceous. All the other cases, however, were decidedly against the phosphorus treatment; whereas, in the case of the simple farinaceous diet we found only four cases out of twenty-four that could fairly be considered as unfavourable.

On a review of the whole evidence furnished by these observations, I think, after making due allowance for all circumstances likely to tell in the opposite direction, that there are fair grounds for the conclusion that a farinaceous diet is likely to be more useful in the treatment of epilepsy than a nitrogenous; but in order to get satisfactory results, observations would have to be extended over a longer period than was at my disposal in the present instance. I hope, however, to continue these observations, and at some future time may record the results of my further experience.

LABYRINTHINE VERTIGO.

MENIÈRE'S DISEASE.

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THE following case, which recently came under my obser-
vation, presents some features of special interest in refer-
ence to the pathology of Menière's disease, a subject now
attracting considerable attention, and so ably handled by
such masters in nerve pathology as Charcot, Hughlings
Jackson, Bourneville, and others.

This case, along with one or two other cases of the same
disease which have come under my notice, serves as the text
for a few considerations on the pathology of this affection.

J. A., ætat. c. 30, a member of the medical profession, engaged in country
practice, consulted me a few months ago in reference to certain distressing
symptoms from which he had repeatedly suffered within the last year or two,
and which had excited in his mind some alarm lest he should be suffering
from brain disease or some form of epilepsy. Occasionally while walking
along the road, or otherwise engaged, he would be suddenly seized with
giddiness and faintness, and a feeling as if he was being whirled to the
right. He never actually fell, as he always succeeded in laying hold of
some support, so as to counteract the seeming rotation. During the attack
external objects seemed to be whirling to tho left. The vertiginous attack
coincided with a buzzing or ringing noise in the left ear.

The patient is otherwise in fair health, but complains of pain in the
region of the left ear, and a ringing sound which varies in degree of
intensity. On otoscopic examination, the left auditory meatus was found to
be somewhat congested and catarrhal, but was not stopped up, nor was
there any discharge. The membrana tympani was also somewhat congested.
The sense of hearing as regards the aerial vibrations of the tuning fork and
to musical notes was unimpaired, and even more acute in the left ear than
in the right. There was, however, complete insensibility in the left ear to

the vibrations of the tuning fork placed on the bridge of the nose or on any part of the skull. These were only perceptible on the right side, in whatever position the tuning fork was placed.

Such briefly were the most prominent symptoms in this case, which I at once diagnosed to be a case of Menière's disease, or disease of the semicircular canals on the left side.

The differentiation of labyrinthine vertigo as a special disease is a brilliant example among many others which might be cited of the application of the results of physiological experiment to the elucidation of disease. It was the remarkable experiments of Flourens on the semicircular canals of pigeons and rabbits which led Menière to associate certain symptoms which he had frequent opportunities of observing in the Deaf and Dumb Institution, of which he was director, with disease of the labyrinth in man; a view which was thoroughly justified by post mortem examination. Flourens found that strange disturbances of equilibrium followed section of the membranous canals of the labyrinth; the character of the phenomena varying with the seat of the lesion. Thus when he divided the horizontal canals, the animal moved its head rapidly from side to side in a horizontal plane, and tended to spin round on a vertical axis. When the posterior vertical canals were divided, the animal made rapid movements of its head backwards and forwards, the eyes were thrown into oscillations, and the animal tended to describe a somersault from before backwards (*culbute en arrière.*) On section of the superior vertical canals, similar phenomena were manifested, but in a reverse direction, the head being rapidly moved from behind forwards and the animal tending ' *culbuter en avant.*'

Combined section of the various canals gave rise to the most complicated and *bizarre* contortions of the head and body.

These disturbances were always most marked when the animal was disturbed, or attempted to make voluntary movements. If left in perfect quietness the disorders of equilibrium gradually subsided though the head was kept in a very abnormal position. When the lesion was limited to one side, the disorders of equilibrium, at first tolerably well marked, ultimately subsided or altogether disappeared. Flourens

kept animals so operated on for long periods of time without observing any cessation of these remarkable phenomena. The animals continued to perform all their usual functions; they could eat and drink, they retained all their special senses, and had not lost the sense of hearing; only the distortion of the head, and the disorders of equilibrium on the slightest movement or disturbance continued as before.

These experiments have been repeated by many investigators, among others, by Vulpian, Harless, Czermak, Brown-Sequard, Goltz, &c., with a view to determine the causation of the strange phenomena. Brown-Sequard and Vulpian attribute the results to irritation caused by the mechanical injuries, and regard them, therefore, as of the character of reflexly excited movements. This view receives support from the fact which Flourens himself ascertained, viz., that movements of the head and body also resulted from '*piqure*,' or mechanical irritation of the membranous canals; movements which, however, subsided when the effects of irritation had passed off. Results in all respects similar to these were likewise shown by Brown-Sequard to follow section of the auditory nerve within the skull. As this operation, however, might cause injury to encephalic centres, it would not be convincing, unless the section of this nerve could be effected without danger of any such complication. Goltz, however, has removed this source of fallacy and confirmed Brown-Sequard's experiment, by removing the whole labyrinth from frogs—an operation equivalent to entire section of the auditory nerve, and capable of being effected without disturbing the contents of the cranial cavity. Frogs so operated on, likewise exhibited disorders of equilibrium in their movements. But the theory of irritation will hardly account for the fact which Flourens' and Goltz's experiments conclusively demonstrate, viz., that the disturbances of equilibrium continue long after the wounds caused by the operative procedure have entirely healed up and become cicatrised. If irritation formed part of the causation of the phenomena following immediately on the operation, it can scarcely be regarded as the efficient cause of their indefinite continuance. Some other explanation has

therefore to be sought for, and evidently in the direction of loss or disharmony of certain impressions by which the functions of equilibration and co-ordination are regulated. Goltz regards the semicircular canals as the origin of impressions which regulate the equilibrium of the head and with it of the whole body. (' *Sie sind, so zu sagen, Sinnesorgane für das Gleichgewicht des Kopfes und mittelbar des ganzen Körpers.*') Let us inquire into the mechanism of these impressions. The power of equilibration and co-ordination of movements is retained by animals deprived of their cerebral hemispheres, and possessing, therefore, no centres above the *corpora quadrigemina* or optic lobes. This can only be experimentally demonstrated in such animals as the frog, the pigeon, the rabbit, in which there is less solidarity among the various encephalic centres than obtains among the higher animals, and specially in man and monkeys. Flourens found that in pigeons previously deprived of their hemispheres the faculty of equilibration became lost after section of the semicircular canals just as in pigeons retaining their cerebral hemispheres. The mechanism of equilibration would therefore come under the head of what are usually termed sensorimotor actions. I shall not here enter into the question as to whether these phenomena imply consciousness, but assume that certain impressions on certain organs of sense are directly correlated and co-ordinated with certain adaptive and responsive actions, phenomena which may rise into consciousness and which under certain abnormal conditions excite painful consciousness.

For a so-called sensori-motor action the integrity of three factors is necessary, viz. : 1. Certain afferent or sensory apparatus. 2. A co-ordinating centre. 3. Efferent or motor tracts which connect the centre with the muscular apparatus specially concerned in each action. A lesion or perversion of either of these three factors causes perturbation of the whole mechanism.

There is considerable analogy between the mechanism of equilibration and that involved in the maintenance of the tone of muscles. The tone of muscles, as Brondgeest has shown, also involves the activity of three factors, viz., peri-

pherical impressions conveyed by afferent nerves, a nerve centre, and the motor nerves of the muscles. The tone is diminished or annihilated by section of the posterior roots or trunks of the sensory nerves, by destruction of the spinal centre, or by division of the motor nerves. In bilateral or antagonistic muscles the loss of tone in one group gives rise to flexor or lateral distortion, or generally distortion in the direction of the unopposed force. In like manner lesion of the sensory, central, or motor part of the mechanism concerned in equilibration will cause, according to the character and extent of the lesion, either complete loss of the faculty or perturbation, which will show itself as reeling, staggering, or rotation, &c.

With the central and motor factors in this mechanism I shall not here concern myself, as I propose to treat this subject elsewhere, but confine my remarks to that factor, disturbance of which is the essential feature of Menière's disease.

The sensory impressions on which equilibration depends are mainly of three kinds, viz., tactile, visual, and labyrinthine. On the harmony or consensus of these three normal equilibration depends, and the perversion or interference with either group causes perturbation of equilibrium, or induces a disharmony which manifests itself in consciousness as vertigo. When tactile impressions are abolished as by section of cutaneous nerves, or by lesions of sensory tracts, equilibration becomes difficult or impossible. In some degree the absence of these may be compensated for by visual impressions, but when the visual impressions are also withdrawn, the faculty of equilibration is entirely annihilated. In the presence of tactile impressions visual are not so necessary, but visual impressions render equilibration more precise, and disturbances of visual impressions, such as by sudden lesions of the eye or abnormal positions of the eye-ball, as result from paralysis of certain ocular muscles, cause manifest perturbations of equilibrium, and give rise to vertigo. Nothing, however, compensates for entire loss of labyrinthine impressions. Visual and tactile impressions may continue normal, but never replace those coming from

the labyrinth, hence pigeons with their semicircular canals destroyed on both sides never recover the faculty of equilibration. It is, however, to be noted that the loss of labyrinthine impressions on one side only does not cause permanent affection of equilibration, the system, as it were, accustoming itself to rely on one set alone. This is a fact of no small importance in reference to the pathology of Menière's disease. The nerve which conveys labyrinthine impressions is not that concerned in the transmission of aerial vibrations. This was shown by Flourens, and has been confirmed by Goltz. These observers found that after destruction of the semicircular canals, animals continued to react even more acutely than before to sounds. Destruction of the cochlea caused deafness, but not disturbance of equilibrium. Hence the nerve supplying the labyrinth, though included in the auditory nerve, is not the nerve concerned in the reception or transmission of sound or aerial vibrations. The nerve which distributes itself to the ampullæ of the membranous canals is the vestibular division of the auditory nerve, which likewise supplies the utricle and saccule. This division of the auditory nerve is the path of transmission of the labyrinthine impressions concerned in the function of equilibration. As to the nature of these impressions, the researches of Goltz, Mach, Breuer, and Crum-Brown have supplied us with much important information.

The membranous canals are supplied with nerves only at their ampullary dilatations. These canals being filled with fluid, it is easy to see that variation in the position of the head will cause different degrees of tension or pressure on the respective ampullæ, and hence impressions may be generated in this manner on the ampullary nerves apart from any impression originating from without.

If we suppose that the head is perfectly still, then the labyrinthine impressions are in a state of statical equilibrium. A movement of the head to the one side or the other will alter these conditions. A sudden deviation of the head will cause simultaneously a plus and minus variation in opposite ampullæ, variations which call forth adapted movements of equilibration. Crum-Brown, who in the main agrees with

Mach and Breuer, has given a most ingenious explanation
of how variations in the tension of the respective ampullæ,
or relative flow of the endolymph, minister to the sense of
rotation, its axis, rate, and direction, apart from visual or
tactile impressions.

If the body be placed on a disc capable of being rotated,
and if the eyes be shut, we are still able to determine the sense
and extent of the angle of rotation. After rotation has been
kept up for some time, the rate gradually diminishes, and
after a time all sense of rotation ceases. If the rotation is
stopped, the individual feels as if he were being whirled round
in the opposite direction. If he opens his eyes, the discord
between the labyrinthine and visual impressions generates a
distressing feeling of vertigo. This feeling of vertigo or
giddiness is also experienced if, while the complementary
rotation is going on, the head is moved so that the axis of
the rotation is no longer vertical. The impression of rota-
tion is generated by the fact that rotation in an axis per-
pendicular to the plane of any of the canals causes the endo-
lymph, on account of its inertia and friction, to press on and
irritate the ampullary nerves in a reverse direction to that of
the rotation. This gradually subsides as the motion of the
canal and its contents become equalised, and ceases altogether
when equalisation is complete. If now the rotation be
stopped, the fluid continues to move on as before in the
direction communicated to it, and so generates a sense of
rotation in the opposite direction, which also ceases when
the fluid comes to a standstill. Crum-Brown thus accounts
for these phenomena :—

'Each canal has an ampulla at one end only, and there
is thus a physical difference between rotation with the am-
pulla first and the rotation with the ampulla last ; and we
can easily suppose the action to be such that only one of
these rotations (say, with the ampulla first, in which case, of
course, there is a flow from the ampulla into the canal) will
affect the nerve terminations at all. One canal can, there-
fore, on this supposition, be affected by, and transmit the
sensation of rotation *about one axis in one direction* only ; and
for complete perception of rotation in any direction about

any axis, *six* semicircular canals are required, in three pairs, each pair having its two canals parallel (or in the same plane), and with their ampullæ turned opposite ways.   Each pair would thus be sensitive to any rotation about a line at right angles to its plane or planes, the one canal being influenced by rotation in the one direction, the other by rotation in the opposition direction.

'Now we have six semicircular canals, three in one ear and three in the other, and I find in all the animals that I have examined, that the exterior canal of one ear is very nearly in the same plane as that of the other ; while the superior canal of one ear is nearly parallel to the posterior canal of the other. . . .

'In each ear there is one canal (the exterior) in a plane at right angles to the mesial plane, and two other canals (the superior and posterior) in planes equally inclined to the mesial plane.' ('Journal of Anatomy and Physiology,' May 1874).   By this arrangement is provided the condition of two oppositely turned ampullæ in a plane at right angles to each axis. We have thus apparently within ourselves a complex self-adjusting mechanism whereby, when the body is at rest, a statical equilibrium is maintained, and in which, by movements of the head and body, impressions are generated by respective variations in tension in opposed ampullæ, which serve not only to indicate the direction of movements, but by the symmetrical plus and minus variations, excite the appropriate bodily movements for maintaining the balance, each inclination in one direction being the stimulus to compensatory action in the other.

Hence we can understand that the balance will be overthrown if one of the forces is withdrawn or abnormally altered.   The perturbation of equilibrium will be in the direction of the unopposed or predominant force.   Now it would appear, from the researches of Flourens, that the horizontal canals mutually oppose each other, and that the superior vertical canals oppose the posterior.   When the horizontal canal on the left side is destroyed, there is a deviation of the head to the left side, and a tendency to spin round from right to left ; so when the superior vertical canals are destroyed,

the equilibrium is overthrown forwards; and when the posterior canals are destroyed, the equilibrium is overthrown backwards. These three directions are the only possible simple axes, but we can readily conceive that the line of movement will be the resultant of the forces which remain, and that this may give rise to exceedingly *bizarre* bodily distortions. If, instead of withdrawing one of the forces, we increase its amount, such as would be the result of irritation, we should expect to find the equilibrium overthrown in exactly the opposite direction to that which would follow from negation.

Hence, if this supposition be correct, abnormal irritation of the left horizontal canal should cause disturbance of the equilibrium towards the right side, and *cœteris paribus* in the case of the others according to the seat of irritation.

Let us now see what bearing these experimental data have on the pathology of Menière's disease.

The characteristic symptoms of the disease are, that the patient is suddenly seized with vertigo and a feeling of nausea or positive sickness, with great constitutional depression and faintness. Usually the giddiness comes on simultaneously with ringing or buzzing in one, or it may be both ears; and generally in the ear principally affected, more or less complete deafness is found to exist, which ultimately becomes absolute.

From what we have already said regarding the results of experimental lesions of the semicircular canals in animals, the general fact of reeling in case of diseased conditions of these structures in man is not difficult to account for; but the point to which I would wish to direct special attention is one which, though of vast importance, not only in relation to this special affection, but in reference to the whole mechanism of equilibration and the action of the co-ordinating centres, has not received that attention which is desirable, viz., the exact character of the reel and the direction in which the equilibrium is overthrown. For on this will depend the conclusion as to the exact nature and seat of the lesion, and considerable light may be thrown on the mechanism of central co-ordination. Now we have seen that

perturbations of equilibrium may result from two distinct forms of lesion of the semicircular canals,—viz., irritative and destructive lesions; and we have seen reason to believe that the phenomena in the one case will be exactly the reverse of the other. To which of these forms does the affection in Menière's disease belong? It is not necessary, as Hughlings Jackson observes, that there should be direct lesion of the semicircular canals in order to produce vertigo and reeling, for indirect affections of the membranous labyrinth, such as result from pressure on the vestibule by foreign bodies, or by syringing the ear, especially in cases of ruptured tympanum, or by the products of inflammatory affections of the meatus, may all cause vertigo and reeling. In these cases we have evidently to deal with irritative lesions, and it would be important to note the direction of reeling in such cases. Usually, I believe, it is towards the opposite side.

Charcot calls special attention to the fact that the vertiginous symptoms and the buzzing in the ear go hand-in-hand. And it is also of importance that the two disappear together, this condition generally coinciding with absolute deafness. These facts indicate that the phenomena are due to irritation, and the paroxysmal character of the attacks renders this still more certain. We have seen that in animals the disturbances of equilibrium consequent on destruction or negation of the labyrinthine impressions on the one side cease after a time, owing to compensation having taken place. This quite coincides with the clinical fact that when the sounds altogether cease the symptoms disappear. The cessation of the sounds and the absolute deafness indicate that not merely the canals but also the cochlea have been invaded by the lesion, which is now negative rather than positive in character.

From the researches of Flourens it would seem that negation of the horizontal canal on one side causes the body to be thrown or spun towards the same side, and that negation of the superior canals causes the body to be thrown forwards, and a like negation of the posterior canals causes the body to be thrown backwards. We should therefore expect that if the lesion is an irritative one the phenomena

would be reversed, and that therefore irritation of the left horizontal canal should cause a feeling of rotation to the right side, and *vice versâ* in the case of irritation of the right; and similarly a feeling of being hurled forwards or backwards according as the irritation affected the posterior or superior canals respectively.

In a case carefully observed by Charcot, in which the lesion principally affected the left ear, the direction of reeling was principally forwards, sometimes it was backwards, and occasionally there was a sense of rotation in a vertical axis *always from left to right.* These attacks always coincided with increased buzzing in the ear; such also was the character of the phenomena in the case I have recorded. In another case which has just come under my care there is also affection of the left ear characterised by paroxysmal buzzing and reeling not dependent on affection of the tympanum.

The direction of the reel is usually to the right side, but frequently the patient feels as if he were suddenly lifted off the ground and pitched forward and to the right side. On one occasion he spun round several times from left to right and fell heavily on the right side of his head, causing a deep scalp wound. This patient always endeavours to keep a considerable distance from the wall when walking with the wall on his right hand, and when he has the wall on the left he keeps near it so as to avoid being pitched over the kerb.

The sense of rotation or actual rotation to the right side is, according to Mach and Brown, caused by irritation of the left horizontal ampulla. Rotation to the right coincides with irritation of the left horizontal ampulla; rotation forwards round a horizontal axis coincides with irritation of the posterior canals, and rotation backwards coincides with irritation of the superior canal. Conversely irritation of these different structures, such as we suppose to be the case in Menière's disease, causes the feeling of rotation to the right, forwards or backwards, according as the lesion affects the left horizontal, posterior, or superior canal respectively. In the normal condition the balance is maintained by the fact that the irritation which causes a tendency to rotation in one direction excites the antagonistic muscles of the body to compensatory action. Hence, for

example, the tendency to backward rotation caused by irritation of the ampullæ of the superior canals, excites reflexly the muscles which tend to pull the body forwards, and so the balance is recovered. In abnormal irritation of one set, however, the compensatory action is insufficient, and so the balance is overthrown. The left superior and right inferior antagonise the right superior and the left inferior, and the left horizontal antagonises the corresponding canal on the right. Now if one set is removed the tendency will be to fall in the direction of the action of the unopposed canals. Hence removal of the posterior canals will cause a tendency to fall backwards in the direction of the influence exerted by the unopposed superior vertical canals. This tendency may, however, be antagonised by voluntary effort in the absence of the reflex mechanism, and so the individual may be able to maintain a condition of stationary equilibrium. This stationary equilibrium, however, is overthrown by any disturbance which draws away the attention, or when the individual attempts other voluntary efforts. This is exactly what is seen in Flourens' experiments, the disorders of equilibrium being always most marked when the animal was disturbed or attempted to move from its place.

Next, in regard to the feeling of giddiness or vertigo. Disturbances of equilibrium do not necessarily cause a feeling of vertigo, and, as Hughlings Jackson justly remarks, it is erroneous to say that the feeling of vertigo is the cause of the reeling. Vertigo is rather the subjective side of perturbed or incongruent sensori-motor co-ordination. In Menière's disease it is chiefly the incongruence between labyrinthine and visual impressions which causes the feeling of giddiness. When the body is rotated round on a revolving disc there is no feeling of giddiness so long as the eyes are shut, as Crum-Brown has shown. If the eyes are opened, however, either during the primary rotation or during the sense of complementary rotation after the disc has ceased to revolve, a feeling of giddiness at once comes on. External objects seem to be revolving also, and this causes a feeling of insecurity or giddiness, and giddiness is also felt if external objects turn round though the individual does not. Labyrinthine

impressions are correlated with ocular movements in the co-ordinating centres, and Flourens' experiments show that oscillation of the eyeballs occurred when the semicircular canals were injured. This is a fact of considerable importance in reference to the mechanism of central co-ordination, and the condition of the eyeballs in attacks of Menière's disease would be well worthy of study. In the case before us there was, along with the feeling of rotation to the right side, a sense of external objects whirling past in the direction from right to left. This is precisely the direction in which objects seem to whirl when the body has been rotated rapidly from left to right. If the eyes are moved rapidly to the right without any rotation of the head or body, the same appearance of movement of objects to the left is caused. This can be easily demonstrated by closure of the left eye, and fixation of the right eye on some distant object. If then the inner side of the right eyeball is pressed on with the finger, the object will appear to move rapidly to the left. Now it has been found by Breuer that when the body has been rotated rapidly for some time to the right, the eyeballs are directed to the right side. Hence it is to be argued that, in cases where from irritation of the left horizontal canal the body appears to be rotated to the right, at the same time the eyes are directed to the right side. This would account for the symptoms in the above case. In like manner I should conclude, where from irritation of the left superior canal the body appears to be rotated backwards and to the right, that the eyeballs will be directed upwards and to the right, and downwards and to the right if the irritation affects the left inferior canal. The direction will be to the left if the lesion affects the corresponding canals on the right.

The next most prominent symptom of Menière's disease is the nausea or sickness, with its accompanying systemic depression.

There is a remarkable relation between vertigo and visceral disturbances. Hughlings Jackson, in a series of most interesting and suggestive speculations, grounds this connection on the intimate relation between the auditory and vagus nuclei in the medulla oblongata, as demonstrated by Lock-

hart Clarke :—' Just as we suppose the tripartite division of
the auditory nerve for the semicircular canals to be afferent
to locomotor centres in the cerebellum, so we suppose the
cochlear division of the auditory nerve to be afferent to
centres for movement of the heart (vascular system generally).
These relations show how it is that hearing is the more emo-
tional, and also the more subjective of the two important
special senses. It explains the cheering effect of music, as
well as accounts for the general disturbances which harsh
noises produce. The auditory is a sense closely akin to the
systemic sensation, to the *Gemeingefühl*. It is significant
that experimental lesion near the auditory nucleus produces
glycosuria by disordering the functions of the liver. Shy-
lock's remarks on the effects of bagpipes on some people
will occur to most in this connection. There is the practice
grooms have of whistling to horses to make them pass
water.' (' Med. Times and Gazette,' August 7, 1875.) In-
teresting lines of inquiry are indicated in these ingenious
speculations.

I am inclined to think that there is a higher co-ordina-
tion of visceral with auditory and other impressions than
that of the medullary nuclei. There is much in favour of the
supposition that visceral impressions form one of the factors
in the consensus which regulates equilibration and co-ordina-
tion, and that therefore their afferent nerves are co-
ordinated with the tactile, optic, and labyrinthine nerves in
the encephalic centres of equilibration and co-ordination.
Czermak found that section of the semicircular canals in
pigeons caused not only the disturbances of equilibrium and
agitation of the eyeballs described by Flourens, but
also very frequently nausea and vomiting. Here therefore
we have another point of similarity betweeen Menière's
disease and experimental lesion of the semicircular canals in
animals. We might infer that the same irritation which,
propagated to the centre, causes ocular disturbances, should
cause visceral disturbances and vomiting if the visceral nerves
were in relation to the same centre. That they are so is
strongly supported by the fact that in certain visceral disturb-
ances we have a distinct form of vertigo—*vertigo a stomacho*

læso of Trousseau, a condition more satisfactorily accounted for
by this relation than by mere disturbances of the circulation.
Further, it is not without significance that animals—the
Felidæ—which have exquisite powers of equilibration and
co-ordination of movements, have large numbers of Pacinian
corpuscles in their mesenteric plexuses, organs which probably
serve in this situation to convey to the encephalic centres the
conditions of pressure in their viscera. These structures are
also found, though not to the same extent, in the mesenteric
plexuses of man.

Visceral disturbances, whether causing vertigo or not,
usually excite nausea and vomiting, and so conversely dis-
turbances of the mechanism of equilibration, either from
central disease or from propagation of irritation affecting the
other afferent factors of this mechanism, such as lesions of
the semicircular canals, may manifest themselves, as reeling
or staggering on the motor side, and as vomiting and its ac-
companiments on the visceral side.

The last symptom of note in Menière's disease is the local
condition of the ear, and particularly as regards hearing.
There is frequently pain which is intensified by sound in
the region of the affected ear. This is quite in accordance
with Flourens' results. He found that injury to the semi-
circular canals caused manifest signs of pain, and that
sounds seemed to cause painful sensation. Hearing is pro-
gressively abolished in the ear affected with the lesion, which
gives rise to the special symptoms of labyrinthine vertigo.
As injury to the semicircular canals did not abolish the
sense of hearing in Flourens' experiments, but as it was
abolished by destruction of the cochlea, we must conclude
that when in the progress of Menière's disease deafness
ensues, the lesion has extended into the cochlea. The sub-
jective sensations of whizzing, ringing, &c., in the affected ear,
will coincide with a stage of abnormal irritation preceding
total annihilation of the function of the auditory nerve.

Usually in Menière's disease deafness is well marked
before the recurrent irritation of the semicircular canals
has ceased to manifest itself in paroxysmal attacks of vertigo
and reeling. The deafness is shown to be of labyrinthine

origin by the insensibility both to the aërial and skull vibrations of the tuning-fork. In the case before us, however, there was a peculiarity which I am not aware of having been previously noticed, and which would seem to indicate a fact of considerable importance in reference to the functions of the semicircular canals in relation to hearing. The patient retained the sensitiveness to the aërial vibrations of the tuning-fork even more acutely than before on the left side than on the right. Not only so, but the power of discrimination of musical sounds seemed sharper on the affected ear than on the other. This I tested by the individual notes and chords within a compass of six octaves on the pianoforte, alternately with the right and left ear, one being closed. The patient was of opinion that the left ear was the more acute.

On the other hand, there was absolute insensibility on the left side to the vibrations of the tuning-fork placed on the bridge of the nose or on any part of the skull. In one or two other cases I have seen, there was complete insensibility both to aërial and skull vibrations on the affected side. The patient who more recently came under my notice, and to whom I have already alluded (p. 34), had complete insensibility to skull vibrations in the left ear; but he stated that he could perceive, though faintly, the aërial vibrations of the tuning-fork placed close to his ear while the other was stopped. He also heard and answered questions when I spoke close to his left ear while the other was stopped. Perhaps this may be in reality due to the action of the right ear, it being difficult to close this absolutely to the reception of sounds. In this case the meatus was not closed, and the tympanic membrane only exhibited slight congestion at the attachment of the malleus, being otherwise apparently normal. I have not had other opportunities of testing the condition of sensibility to aërial and skull vibrations in Menière's disease. Should the above observations be confirmed, they will serve to show that the semicircular canals are, as regards hearing, specially concerned in the reception and transmission of skull vibrations in contradistinction to aërial vibrations.

ON THE PHYSIOLOGICAL ACTIONS OF HYOSCYAMINE.

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HYOSCYAMUS is an important member of the small but active group of medicines which are sometimes classified under the name of mydriatics or dilators of the pupil. Belladonna, the best known of the group, has been the subject of careful and extensive research, but hyoscyamus, and its active principle hyoscyamine, have not till recently been subjected to such a scientific investigation as to afford physiological grounds for the adoption of opinions which have been arrived at by analogy and experience, regarding the therapeutic value of the drug. For a considerable time it has been believed that the properties of the active principles of belladonna, hyoscyamus, stramonium, and even dulcamara, are analogous, if not identical. But it is not improbable that, although on the whole there are many actions which are common to all the members of the group, it may yet be shown that their active principles are not identical, inasmuch as they vary, in the intensity at least, of the symptoms which they produce. Thus there can be no doubt that hyoscyamus is more decidedly and directly hypnotic than belladonna. It is also more powerfully and persistently diuretic. It has a more decided action in reducing the bodily temperature, and there is some reason to believe that it acts more rapidly as a

mydriatic, both when locally applied and when administered internally. Yet the properties of the substances are so fully analogous, that the method employed in investigating the actions of the one is that which is most likely to lead to the determination of those of the other. Consequently, the actions of hyoscyamine may to a certain extent be determined by the means employed in demonstrating those of atropia.

The preparation of hyoscyamine which I have used in the following experiments is the amorphous alkaloid. The alkaloid in this form is very readily soluble, and convenient for hypodermic use. The specimens which I have employed were prepared by Messrs. Smith, of Edinburgh, and have been of decided and uniform strength.

It will be convenient to classify the results obtained under the following heads :—

1. The action on animals of small, large, and lethal doses.

2. The effect produced by prolonged administration.

3. The analysis of the physiological action, embracing the effect on special systems and tissues, and on the temperature.

4. The action on man.

5. The therapeutic actions.

First, with regard to the action of small doses. One grain of the alkaloid administered in four minims of alcohol and five of water produces decided symptoms in a cat or rabbit. The most marked of these are dilatation of the pupil, dryness of the mouth and throat, slight numerical depression of the pulse, followed by a sudden and very characteristic rise; steady fall of temperature, sometimes quickening of respiration, great restlessness and apparent delirium, with impairment of motor and sensory function, followed by renewed excitement and recovery. Frequently in rabbits, but rarely in cats, this quantity is sufficient to produce sleep—the animal sinking down in a state of exhaustion after the period of primary excitement. Urine is also frequently voided, but the bowels are rarely moved, and when

they do act it is only for the expulsion of a very scanty and exceedingly dry motion. The first evident physiological change following upon the injection of the drug is dilatation of the pupil. This commences in about two minutes after medication, and advances till in less than a quarter of an hour full dilatation is reached. In cats this phenomenon is very well marked, inasmuch as the pointed oval of the natural pupil, after becoming somewhat irregularly rounded, changes suddenly into a complete and expansive circle. The animal then begins to move about uneasily, and soon rubs the neighbourhood of its mouth and nose with its paws, and on the insertion of clean blotting-paper into the mouth it is found that it and the fauces are absolutely dry, the nose also being dry and hot. During the first half-hour the pulse steadily descends, falling altogether about twenty beats. It then rises to a great height very suddenly, and this ascent is generally simultaneous with the development of excitement and delirium, or the manifestation of the somnambulistic state. In this sudden elevation, the pulse may, in five minutes rise as much as sixty beats, and may move upwards for half an hour, till in that time it has increased one hundred beats a minute. At the same time considerable muscular weakness is present. The animal either crouches down through the inability of the limbs to support the weight of the trunk, or struggling to maintain its natural carriage it inclines to fall upon one or other hind-quarter. The head nods as if the animal were drowsy and the cervical muscles weak. The temperature falls steadily from the beginning, till in the first half-hour the total decline may be about 3°. The respirations when an animal is under the influence of a small dose are of variable character and rapidity. In rabbits they incline to rise, but the increase may be due to the existent state of excitement, and not to any direct action of the medicine. Thus in cats, which are much less influenced by excitement than rabbits, the respirations vary very little under the operation of small doses, but what little divergence there is from the normal index is towards a fall. Should the period of excitement not be followed

by sleep, the pulse remains persistently high for about half an hour. The vascular activity is associated with constant muscular movement ; the animal struggling to rise from the couchant position finds that it cannot support itself. The hind-legs are invariably the seat of the greatest weakness, and the cat or rabbit by acting powerfully with the fore-legs and muscles of the trunk pushes the paralysed limbs backwards, and sometimes by a muscular effort of the forepart of its body almost turns a summersault. These backward movements appear to be due either to some hallucination or to the general impulse to activity, operating in such a manner as to push the body away from the strongest point which acts as a mechanical fulcrum. When sleep does not succeed this period of excitement, the pulse after the lapse of about half an hour falls suddenly to somewhat below the starting-point, and in about ten minutes again rises till the normal index is reached. The mouth becomes somewhat moister, muscular strength is regained, and the animal appears to return to its normal condition. The pupils, however, remain dilated, and may continue so for several days. When the motor paralysis is at its height, there is also considerable sensory deficiency, as pinching and pricking evince little or no movement, and the respirations are not heightened under irritation. The accompanying table will show the course of the pulse and respirations in such a case as that just described. The temperature was not taken in the first instance, in order that the changes in the cardiac and respiratory functions might be observed without the production of fallacy by extraneous irritation. The same dose, however, was given to another cat of about the same weight, and the pulse followed a similar course. In this case the temperature was specially observed, and is embodied in the same table. It will be seen that the fall of temperature was uninterrupted, and that there was no rise simultaneously with the return of normal cardiac action, but in numerous instances it was observed that the temperature had almost risen to the starting-point on the day after the experiment.

TABLE I.

Showing the influence of small doses of Hyoscyamine on the pulsations, respirations, and temperature of a Cat.

Time	Pulsations	Respirations	Temperature
3·5	184	74	100·4°
(Hyoscyam. 1 grain.)	—	—	—
3·10	176	44	99·8
3·15	172	50	99·3
3·20	168	58	98·8
3·25	168	58	98·6
3·30	168	60	98·5
3·35	164	65	98
3·40	225	55	98
3·42	224	41	98
3·45	224	50	97·5
3·50	229	58	97
3·52	236	55	97
3·54	236	54	97
3·58	264	52	97
4·2	261	49	97
4·6	240	53	96·8
4·10	240	55	96·8
4·12	190	59	96·7
4·15	182	55	96·7
4·20	180	51	96·6
4·25	186	55	96·6

When still smaller doses are administered, the results are characteristically modified. Thus half a grain was injected in solution under the skin of a rabbit weighing thirty-four ounces. In half an hour the pulse had fallen forty beats per minute. The pupil was dilated one-fortieth of an inch, or one point on the pupilometer employed, and though there was a temporary rise in pulsation during excitement, it again fell, and never from the beginning to the end of the observation reached the starting-point. The respirations increased in number, and the temperature fell only 1°. It is seen by this that very small doses depress the pulse without subsequent rise, just as it will be found further on that large doses produce increased cardiac action without the preliminary depression which characterises the operation of moderate quantities of the drug.

With regard to the effect of large but not lethal doses of hyoscyamine, the clearest idea of their action will be

obtained from a perusal of the record of symptoms developed by them :—

A cat weighing 6lbs. 10ozs. had a three grain dose of hyoscyamine injected hypodermically. Before the medicine was administered the pulsations of the femoral artery were 150 per minute. The respirations were 42, the temperature 102·4°, and the pupils 4 lines by 2. The drug was given at 11.15 A.M.; at 11.17 the pupils were dilated to their full extent, being half an inch in all diameters. The animal was exceedingly restless. At 11.20 the mouth was dry and glazed. The respiration was much quickened, but still the motor power was unimpaired. At 11.30 it lay down, and when released fell awkwardly from the table, and endeavoured unsuccessfully to jump upon a ledge about three feet from the floor. At 11.45 the pulse was 186, the respirations 176, and the temperature 100°. During examination it struggled a good deal and then fell back exhausted. When placed on the floor it subsided immediately. At 12.0 it attempted to walk, but swayed from side to side, and when irritated into temporary movement fell down heavily as soon as the stimulus was removed. At 12.10 it was irritated mechanically, and with great effort managed to jump upon a chair, but on trying to reach the top of a low table completely failed. Respirations 130, when at rest. At 12.15 there was great motor paralysis, but the animal showed no defect of sensory appreciation. It felt the prick of a needle on the limbs and trunk, and started after stamping or a shrill whistle. Respirations 114, pulsations 180, temperature 99·8°. At one o'clock the muscular system was completely prostrated, and along with the full development of motor paralysis there was a great reduction in the respirations. When the cat struggled it inclined to fall backwards, owing to the great weakness of the hind limbs and the relative activity of the paws. Pulsation 186, respirations 58, temperature 97·2°. At 1.30 the animal still struggled while under examination, but during the excitement the respirations did not increase in number—Respirations 40 (somewhat sighing), pulsations 172, temperature 96·7°. Immediately after this observation prostration became complete. However much the animal was irritated it could not retaliate, though it showed ample signs of being sensible of impressions. At 2.30 there was an occasional spontaneous movement and a sharp angry cry, but between these movements the cat slept soundly. There was great impairment of sensibility even during the short intervals of wakefulness. The mouth was exceedingly dry—Pulsations 184, respirations 24 (diaphragmatic), temperature 93°. At three o'clock sensory paralysis was more marked. The cat could be pinched and pricked severely without giving any sign of feeling. The legs were slightly retracted on irritation. When held up with the limbs clear of the ground they were very feebly moved, but the hind-legs were quite as active in that position as the fore ones. When left alone it slept heavily for short periods, and breathed in a spasmodic manner, taking two or three interrupted inspirations to each expiration. It sometimes woke up and endeavoured to stand, but could never extend its hind-legs. When it failed and succumbed under the effort, it gave a short angry cry. At 3.15 the pupils were still fully dilated and the mouth dry. The animal could be roused to consciousness, and was conscious during the intervals of excite-

ment—Pulsations 184, respirations 22, temperature 91°.   At four o'clock there was great restlessness between snatches of sleep. It attempted to walk, but swayed constantly from side to side, and could not stand erect. At five it was in much the same condition—Pulsations 180, respirations 30, temperature 89·8°. Sensation was best marked inside the ears, but was deficient everywhere.   It heard shrill whistling, and made respondent movements.   When irritated, it pushed itself vigorously backwards, but was unable to stand erect.   Every movement was one of retrogression, and six times it thrust itself backwards more than its own length by the action of the paws, and terminated each effort by nearly completing a backward summersault. At seven o'clock the cat was still very peculiar. It hung the head over the edge of the table in an aimless manner, and then fell into interrupted sleep. It still ran backwards when irritated. There was a good deal of general tremor—Pulsations 180, respirations 30, temperature 90·4°. At eight it was almost well, and walked about deliberately, but with a swaying movement.   The marked improvement in muscular tone was accompanied by a decided increase in the respirations, just as the accession of motor palsy was associated with their fall. At this time the pulse was 141, the respirations 40, and the temperature 91·4°.   Next morning the animal was comparatively active, but very timid. Sensation was completely restored.   The pupils were widely dilated—Pulsations 150, respirations 72, temperature 97°.

It will be seen that in this instance there was no appreciable preliminary fall of pulsation, the dose having been such as to produce almost immediate excitement.   The great decline in temperature is also most noticeable, associated as it is with such a marked increase of cardiac activity.   The relation between the temperature and the respirations is so precise as to suggest an explanation of this anomaly.   The beginning of this precipitate decline in temperature exactly corresponds with that of the slackening of respiration, and when the respirations were at their lowest the temperature reached the minimum of 12·4° below the starting-point. Two influences suggest themselves as active in producing this great reduction of temperature during complete prostration from hyoscyamine.   These are the inactive condition of the muscles which arrests the progress of oxygenation of muscular tissue, and the partial paralysis of respiration which diminishes the supply of oxygen to those and other textures.   A special action upon the pneumogastric, which will be subsequenty referred to, may also have some effect in producing this remarkable fall of temperature, which, occurring in so limited a time as five hours,

is quickly followed by a complete return to the natural index.

Almost exactly similar results followed upon the administration of 5 grains of hyoscyamine in solution to a dog. The following table will be self-explanatory, and will also supply a *resumé* of the previous observations :—

TABLE II.

Changes in pulse, respirations, and temperature of a Dog under a large dose of Hyoscyamine.

Time	Pulsations	Respirations	Temperature
11·30	108	30	102·8°
(5 grains).	—	—	—
12	146	60	102·3
12·5	155	66	102
12·30	176	68	101·4
12·45	180	78	100·4
1	168	34	100·2
1·15	182	18	100
1·30	198	26	100
1·45	178	28	100
2·35	185	34	100.5
2·45	180	32	100·4
3	180	26	101
3·30	170	22	100·4
4·35	170	18	100·2
5·45	155	—	—

Here also it will be seen that the maximum height of the pulse and the lowest index of the respirations and temperature occurred simultaneously; and observations taken during the period of greatest depression show that these indications were reached during the time when the animal was almost comatose. At that time it could be roused only with great difficulty by shrill whistling, loud stamping, and the infliction of bodily pain. Sometimes, however, it awoke spontaneously and struggled to rise, but was incapable of moving any part of the body satisfactorily. After the effort it invariably fell back in an exhausted condition, and sank again into semi-coma. During the periods of wakefulness he manifested a considerable amount of sensory impairment, and in the somnambulistic stage the respirations were of a very peculiar nature. There were three or four hiccupy inspirations to each expiration, and during these spasmodic inhalations the muscles of one or both sides of the face were sometimes slightly twitched, while the head was almost invariably jerked upwards. About three hours from the time of administration of the drug, the period of renewed excitement commenced, and was characterised by a slight and transient rise in the pulse, along with an increase of respirations and temperature. The animal became more restless; the intervals between snatches of sleep were more prolonged, and the

periods of uninterrupted rest were shorter. It then struggled without intermission, trying unsuccessfully to support itself on its limbs. Under the influence of additional irritation it was able to stand erect, but only staggered about for half a minute, then fell down heavily, and presented every appearance of utter exhaustion. It was quite conscious, however, and made responsive movements to such stimuli as it could appreciate. Several times in this stage it passed a small quantity of urine, a few drops of which when applied to a rabbit's eye caused rapid dilatation of the pupil. The improvement in muscular tone was gradual but certain, and nine hours after the injection of the hyoscyamine there were no symptoms of derangement beyond slightly spasmodic inspirations, motor weakness, and a tendency to drowsiness. The pupils were also widely dilated. At 9.30 next morning this last appearance was the only abnormality presented by the animal.

From these and other observations, therefore, it may be fairly inferred that quantities of hyoscyamine, which are almost sufficiently large to cause poisoning, produce rapid and complete dilatation of the pupil, increased pulsation not preceded by an appreciable fall, excitement followed by a somnambulistic condition in which deep sleep intermits with a struggle to overcome the inertia resulting from muscular prostration. This condition is followed by a short return of excitement, accompanied by an increased activity of cardiac and respiratory function, and a greater evolution of animal heat. During this period the motor paralysis disappears, and sensation, which had been somewhat impaired, again becomes normally acute. The pupils continue dilated and the limbs somewhat weak, but beyond these signs, and the existence of a certain amount of drowsiness, the animal manifests no appearance of bodily or mental derangement.

Lethal doses of hyoscyamine produce death, either suddenly during the stage of excitement, or more slowly by exhaustion and coma. In the first group of cases the death is materially hastened by the resistance which the animals offer to restraint during examination in the excited stage. In the second, the animal, after passing through a period of excitement in which the pulse has been exceedingly quick and the respiration also hurried, enters into a semi-comatose condition in which the respirations sink to an abnormal extent, and the pulse becomes absolutely imperceptible. The coma continues, the respirations fall still lower, and suddenly the pulse again becomes perceptible. On its reappearance it

is almost reduced to its normal index, but has an unnatural strength and precision of impulse. Very soon afterwards the respiration becomes slow and gasping, the cardiac action gets gradually imperceptible, and the animal dies.

An illustration of the first mode of action of lethal doses is supplied by the case of a rabbit poisoned in fifty-five minutes by two grains of hyoscyamine.

The rabbit weighed 2lbs. 15ozs. Previous to the administration of the drug the respirations were 60, the pulsations 146, the pupils 12½ points on the pupilometer, and the temperature 100·4°. The pupils almost immediately began to dilate, and in twenty minutes measured 15 points. At 10.30, half an hour after the injection, the pulse was 180, and the temperature unchanged. The respirations were quickened and of an excited character. Motor power was much diminished in the fore-legs, but sensation was everywhere intact. The animal was quite conscious, but when irritated could not move away from the source of irritation. The body was exceedingly tremulous, and towards the end of the observation there was an inclination to sleep. At 10.45 the respirations were 114, the pulsations 224, and the temperature 99·9°. There was now great muscular prostration. The head lay flat upon the table, and though conscious of impressions, both through the special and general senses, the rabbit was utterly unable to move. When held with its legs clear of the floor it moved them only slightly, and for a very short time. When placed on its side it was able to roll round upon its belly after a severe struggle, which caused almost fatal dyspnœa. At 11.0 the pulsations were 285, the respirations 78, the pupils 15 points, and the temperature 97·7°. The rabbit struggled violently while under examination, and died during the thermometrical observation. Large quantities of urine were twice excreted.

This last circumstance throws some light upon the fact that there are two definite modes of death from large doses of hyoscyamine. In the case of animals dying suddenly, while the pulse is excessively high and the muscular power sufficient to admit of a considerable amount of struggling, there is invariably a large secretion of urine and little narcotic action. In such cases death results from syncope but when muscular activity is not excited by external irritation, and when there is no secretion of urine, head symptoms develop with marked rapidity and intensity; sleep, at first interrupted by spontaneous startings and short periods of delirious excitement, passes into absolute coma, and death ensues after a temporary period of renewed cardiac excitement. In the first class of cases the brain is found to be somewhat anæmic, and little or no blood escapes on its re-

moval. The bladder is generally full and distended, and the right and left sides of the heart contain some fluid blood. In the second, the cavity of the skull is full of fluid blood, large quantities escaping from the sinuses. The medulla and cerebellum are surrounded by venous fluid, and the surface of the brain is marked by large vessels, though the cerebral substance is pale and anæmic. The corpora striata appear in such instances to contain relatively more blood than any other part of the brain. The heart and lungs are full of fluid blood, and the latter also edematous. The kidneys are always congested. These observations will be illustrated by the case of a cat to which a poisonous dose of hyoscyamine was administered.

The following observations were recorded prior to the hypodermic injection of five grains of the amorphous alkaloid :—Pulsations 144, respirations 31, temperature 101·8°, pupils 4 points by 2, weight 6lbs. 4ozs. During this examination it passed urine. The conjunctivæ were somewhat injected. The drug was given at 10.35. At 10.40 the pupils were almost fully dilated, and the animal was very excited. At 10.45 it was exceedingly restless, and struggled vigorously—Pulsations 224, respirations 50, temperature 100·6°, pupils fully dilated. At 11.0, it was weak, but still very much excited—Pulsations 236, respirations 50, temperature 100·4°. At 11.15 it still struggled and had shown no inclination to sleep. Pulsations 240, respirations 60, temperature 99·5°. There was no more injection of the eyeballs than at the commencement. At 11.50 the cat was asleep, the pulse had become imperceptible, the respirations were 40, and the temperature 98·6°. When placed on the floor, it lay upon its belly with its fore and hind legs stretched out in a sprawling manner. After lying for some time it commenced spontaneously to struggle, and with great difficulty succeeded in rising. It walked for a few steps in a feeble and tottering manner. Its mouth was very dry and parched. At 12.0 the femoral and cardiac impulse was quite inappreciable. It slept heavily, but started at intervals without external stimulation, and after a moment or two was again prostrated by sleep and muscular weakness. When sufficiently roused, it was quite conscious and made efforts to rise during shrill whistling or loud stamping on the floor. Respirations 36, temperature 96·8°. The slightest struggle at this time caused great dyspnœa. When the tail was pinched the animal attempted to raise its head and made an effort to cry, but was only able to give a feeble gasp. There was apparent defect of sensibility to pain, most marked in the hind-quarters. At 12.15 it was quite comatose, and could not be roused by any amount of irritation. Respirations 36, regular. There was no increased injection of the conjunctivæ. At 12.25 there was continued coma, but the limbs and tail were sometimes moved spontaneously, and the movements repeated in an automatic manner. At 12.40 the pulse again became perceptible and measured 168. It was somewhat intermittent and characterised by numerous variations of arterial pressure. At the same time there were occasional twitchings of the face, ears, and fore-legs. The

coma was profound. The tongue, the hard and soft palate, and the gums and nose were dry and hot—Temperature 93·6°. *No urine had been passed since the commencement of the observation.* At 12.50 the cat could not be roused. The mouth was absolutely dry and blood oozed from the gums. The pupils were fully dilated and the cornea quite insensible. There were occasional spontaneous but feeble movements of the legs and head. Respirations 24 (shallow), pulsations 180. At 1.0 the same condition of prostration continued—Pulsations 170, respirations 13. At 1.7 there were rapid movements of a vermiform character, due to the excitement of an intra-uterine kitten which came to life just as the cat died, and which on removal after death, was brought round by artificial respiration and lived for more than three hours.

The *post mortem* appearances observed in this case were characteristic only as far as the thoracic and abdominal cavities were concerned, on account of the sections required for emptying the uterus and the draining away of blood during a prolonged examination of the movements of the cardiac walls. The operation was performed very shortly after death, and on exposure of the heart the auricles were making 42 contractions per minute. There was no perceptible appearance of spontaneous contraction of the ventricles. The diaphragm made, without any irritation, five slight contractions a minute. At 1.30 the auricular contractions had diminished to 36. At 2.10 the right auricle acted 24 times, and the left 7 times per minute. At 2.15 there was very faint and limited contraction of the right ventricle, just at the root of the pulmonary artery, where some fluid blood had accumulated. At 2.20 all movement was stopped, and did not again commence. On opening into the heart *in situ,* it was found that both sides contained fluid blood. The lungs were slightly congested and edematous. The liver and kidneys contained a large quantity of dark blood, and *the bladder was empty.* The condition of the circulation in the cranial and spinal cavities was altered by the prolonged draining from the open abdomen and thorax. They contained no blood, and the brain and spinal cord were anæmic. From the time that the body was opened no peristaltic movements were visible, nor could they be excited artificially. There were no contractions of the uterine fibres, even under irritation.

These observations regarding peristalsis were made because it has been advanced by Meuriot that, in poisoning by atropia, the intestines were found on exposure to be undergoing violent contractions, owing to the action of belladonna as an excitant of the non-striated muscles.[1] On the other hand, Bloebaum and Bezold have stated that, as the result of experiment, they have reached the conclusion that on the intestines, bladder, uterus, &c., atropia has a sedative action. They go so far in this direction as to state that the muscular

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[1] On opening into the abdomen of a rabbit twenty minutes after death by atropia poisoning, Dr. Arbuckle, of this Asylum, found the bowels active with spontaneous peristaltic movements.

fibre of these organs may be completely paralysed by that drug.

In no other animal examined immediately after death by hyoscyamine poisoning, were peristaltic movements of the intestines visible.

Other animals were specially inspected for the purpose of determining the state of the intra-cranial circulation in poisoning by large doses of the drug. In a rabbit which had passed no urine whatever during a prolonged observation, there was a large quantity of fluid blood in the sinuses and membranes of the cranium, and also in the spinal cavity. The superficial aspect of the brain was somewhat injected, but on section the substance was pale. The *corpora striata* contained more blood than any other part of the cerebrum. In another cat that died after the administration of a five grain dose there was also a large quantity of fluid blood collected in both the cranial and spinal cavities. The brain substance was pale on section, though somewhat congested externally. The bladder was empty, and no urine had been passed after the administration of the poisonous dose. Both sides of the heart were distended with fluid blood. There were no peristaltic movements of the bowels. The lungs were somewhat congested and edematous.

There can be no doubt that the intra-cranial circulation in instances where the animal reaches death through coma, and where there has been a retention of urine, and consequently an impaired elimination of the poison, is very different from that anæmic state which exists when, though the medicine is being rapidly excreted by the urine, the animal suddenly and unexpectedly falls back in fatal syncope.

Before quitting this part of the subject it is necessary to make a few observations on one important difference which exists between belladonna and hyoscyamus, with regard to the almost complete immunity which some of the lower animals enjoy from the poisoning action of the former. Weight for weight, hyoscyamine is quite as fatal to rabbits and pigeons as to dogs and cats. On pigeons, however, it resembles atropia in having no action on the pupil, either when applied locally or given hypodermically. Neither do fatal doses produce in them dryness of the tongue or palate.

The guinea-pig also is moderately susceptible to the action of hyoscyamine. One grain killed a full-grown animal weighing 38ozs. The same guinea-pig, however, had had an equal dose twenty-four hours previously. Two grains killed in one hour a rabbit weighing 3lbs.; and three grains is an almost certainly fatal dose to these animals. It is probable that a certain amount of tolerance of the drug is acquired, as I have observed, in animals who have twice had moderate quantities of it, that the effect produced on the second occasion was much less marked, even though the second dose was larger than the first. Thus, to a dog which was completely prostrated with five grains, nearly double that quantity was administered in a week afterwards with much less effect. To a rabbit I have given one grain daily for fifteen days without producing anything approaching to a fatal result, and the administration could have been prolonged but for the accession of subcutaneous cellulitis, which might have vitiated the results aimed at. Still there is not sufficient evidence to show that a general tolerance of the substance is established, as the apparent difference in symptoms might be due to its increased and more rapid elimination in one case than in another. Yet there is no doubt that, whereas large quantities of atropia have been administered to rabbits without fatal results, a very small quantity of hyoscyamine is sufficient to cause in them physiological action and even death. Instances of this action have already been given, and others will subsequently be adduced. With regard to pigeons, I have found that though they have been known to recover after the administration of two grains of atropia by hypodermic injection and three grains by the mouth,[1] less than half a grain of hyoscyamine is required to kill a full-grown bird. The symptoms produced are remarkably like those already recorded as occurring in rabbits, cats, and dogs, as the following experiments will show.

One grain of the amorphous alkaloid was injected under the skin of a full-grown pigeon. Previous to the administration, the pupils were accurately measured. In order to avoid all fallacy with regard to the action upon the irides, the pigeons were specially watched in

[1] 'A Treatise on Therapeutics,' Wood, Philadelphia. P. 208.

a darkened room used for ophthalmoscopic purposes, and always examined in the same position and at the same distance from the solitary light. In this case the pupils at their point of fullest dilatation measured 5½ spaces on the pupilometer. At the same time it was noticed that the birds could voluntarily expand and contract the pupils over a limited radius. The walking and flying of the bird were primarily tested, and one grain of hyoscyamine was injected at 3.20 P.M. At 3.25 the drug had already taken so much effect that the pigeon was utterly unable to support itself. It was restless, however, and moved its head incessantly from side to side. The pulse was very quick, and measured at least 300 a minute; and the heart's impulse, which was previously imperceptible, was now quite distinct. The respirations were slow. There was sudden collapse, and the last remaining movement was a fluttering action of the tail. *There was not the slightest dilatation of the pupil.* At 3.26 the pigeon died. The heart, which was examined before the brain, contained fluid blood in both sides. The brain was anæmic. In this case the course of the symptoms was too rapid to admit of satisfactory measurements.

To another full-grown pigeon one-fifteenth of a grain was administered hypodermically. This was done at four o'clock. At 4.6 it seemed somewhat excited, but walked as smartly as before. It ran to a dark corner and cooed sharply, and much more frequently than it had done while under observation earlier in the day. There was no dilatation. At 4.15 it was still active and flew as well as before. The pupils were carefully measured and showed no dilatation. At 4.20 the motor power was still good; but the pigeon never moved spontaneously, and when startled with stamping or other irritation, never walked so far as it had done before. At 4.30 it could still stand erect, and when thrown into the air could fly and land on the floor naturally. At 4.35 the pupils were again measured and showed no dilatation. There was slight strengthening, however, of the cardiac impulse. Pulsations 240, respirations about 60. At 4.45, as the dose did not appear to be producing any effect, an additional fifteenth of a grain was injected in another spot. After the injection it walked off smartly, and persisted in standing upon its legs. At 4.50 it appeared somewhat heavier, but still maintained the standing attitude. At 4.55 the pupil was again measured, but no dilatation whatever was detected. There were appearances of diminished muscular precision and power. The walk of the bird was somewhat embarrassed and heavy. When thrown in the air it flew well, but on reaching the floor could not balance itself without using its wings. It never moved spontaneously. At 5.0 it commenced to crouch down through weakness of the legs. When gently thrown up to fly it came down with a thump, having used its wings very imperfectly. For a short time it lay where it fell, without changing its position. At 5.3 there was great weakness. The pigeon could not balance itself on the observer's arm, as up to this time it had readily done, and on reaching the ground it lay in a crouching attitude. The pupils were again measured with negative results. At 5.5 there was complete prostration. The bird could not rise under any stimulation. It was fully conscious of impressions, and when irritated struggled hard to move away. Pulsations 280, respirations 25. At 5.10 the state of the pupils was again ascertained by measurement, but no dilatation existed. The bird was exceedingly drowsy and helpless, but made

occasional spontaneous movements—raising its tail and flapping its wings. *The mouth was examined and was found to be quite moist.* At 5.15 the pupilometer was again applied, but no dilatation was present. In fact, the voluntary expansions of the pupil were now impaired, and the aperture was consequently contracted instead of increased. The pulse was now completely imperceptible ; the respirations 12 per minute and gasping. There was complete prostration, but sensation was very little impaired, as the least touch caused instant starting. At 5.20, it gave a few convulsive flaps of the wings, and while lying on its side pressed its body backwards. This process was twice repeated. It was again ascertained by measurement that the pupil was fixed at the same size as before. There were a few spontaneous movements of the legs and tail. The pigeon, however, was moribund, and died at 5.23. Immediately after death, the pupils dilated to 7 points on the pupilometer, thus acquiring a diameter of $1\frac{1}{2}$ points more than the extreme extent of dilatation previous to the administration of hyoscyamine.[1]

It will be useful to compare this record with that describing similar cases of poisoning in cats and dogs. In both there is a marked fall of respiratory action and a decided rise in the pulse immediately preceding or accompanying prostration. There is an increase of arterial tension after the administration of the drug, a decrease during the stage of collapse, and again a temporary increase of arterial pressure with slowing of the pulse immediately before death. In the pigeon, as in the cat, this last change was well marked, being in the pigeon a change from 280 to 198, and in the cat from 240 to 168. But if the similarity of the action of hyoscyamine on birds and mammals is interesting, the discrepancy is much more so, inasmuch as the two most perceptible effects—the dryness of the mouth and throat, and the dilatation of the pupils—which characterise the action of the drug on the latter, are altogether wanting in the case of the former. No satisfactory explanation of this variation shown by birds from the ordinary effect of hyoscyamus upon the pupil can at present be given, but there is no doubt that when it is discovered it will be found in that altered anatomical condition which must exist as the cause of the similar inactivity of opium as shown by Dr. S. Weir Mitchell, and of calabar bean as demonstrated by Vée and Leven. Wharton Jones advanced the opinion that the reason for this variation was that the irides of birds had no radiating fibres, but recent anatomical investigation has

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[1] These experiments with pigeons were frequently repeated, and with the same results as those just recorded.

shown that such fibres do exist, and the cause of the pheno-
menon is still problematical.

Effect of prolonged administration.—The prolonged em-
ployment of hyoscyamine results in important modifications
in the action of the drug. A certain amount of tolerance is
established after repeated administration. The temperature
and the respiration become much less affected by single
doses, and the marked loss of weight which occurs for the
first week or more is substituted by a decided improvement
in bodily condition during the subsequent period when
exactly the same quantity of the drug is given. The follow-
ing table will show the effect produced by small doses
regularly repeated :—

TABLE III.

Effect of one grain of Hyoscyamine administered to a Rabbit daily.

Date	P.	R.	T.	Weight	Pupils
			Degrees	Lbs. Ozs.	Points
June 1	138	80	102	3 4	15
,, 2	166	120	103	3	16
,, 3	144	132	103·5	2 14¼	16
,, 4	150	126	104·4	2 15½	16
,, 5	186	136	103·2	2 15¾	16
,, 6	184	135	104·3	2 15	16
,, 7	185	132	103·8	2 12½	16
,, 8	216	121	104	2 11½	16
,, 9	264	124	104·4	2 12	16
,, 10	240	120	103·8	2 14¾	16
,, 11	220	120	104·3	2 13¾	16
,, 12	240	120	104	2 12½	16
,, 13	240	126	104·3	2 12¼	16
,, 14	240	110	104·4	2 14¼	16
,, 15	240	120	104	2 14	16
,, 16	198	126	104	2 13¼	16
,, 17	236	116	104	2 14½	16
,, 18	206	128	103·8	2 15¾	16
,, 19	240	126	104·5	2 15¾	16
,, 20	210	126	103·8	3 2½	16
,, 21	236	126	104·2	3	16
,, 22	264	120	104·2	3 1	16
,, 23	240	124	103·6	3 2¼	16
,, 24	240	124	104	3 2½	16

This record shows that the effect of repeated small doses
is to produce a rise in the pulse to almost double its natural
index ; a similar and almost equal rise in the respirations ; a

persistent elevation of about 2° in temperature, and rapid dilatation of the pupil to its full extent. During the first week there is loss of weight, which is persistent during the second. During the third week flesh begins to be regained, and towards the end of the month the initial weight is almost reached, independent of the constant administration of the drug.

On June 20th, when this rabbit had been for three weeks under the influence of the drug, it was determined to watch how much the actions of individual doses of the medicine might be modified by the continued employment of it. It was found that the pulse followed its usual course, and rose in 1½ hours from 210 to 280, and the respirations also rose from 126 to 140; but the temperature instead of falling about 2°, as it would have done in a fresh rabbit, rose from 103·8° to 104°. In another hour it had reached 104·2°, and in 3½ hours from the time of administration had again fallen to the starting-point. The motor symptoms were like the numerical changes in respiration and pulsation, similar to those produced in a sound rabbit by a small dose of the drug, but there was little or no cerebral excitement, and little or no alteration in the character of the respirations, which were quick and not abdominal. The mouth also was found on examination to be fairly moist.

The inference to be drawn from the record of the physiological changes produced in an animal under the prolonged influence of hyoscyamine, appears to be that after a certain constitutional condition has been produced by the drug, the only action of additional doses is one of stimulation of the sympathetic and depression of the motor nervous system. This appears to me to show that during the action of the medicine on animals treated for the first time, not only the sympathetic but the pneumogastric nerves are affected. Stimulation of the sympathetic would produce the changes in the pulse which result from the use of hyoscyamine, but it would not lead to the associated alterations in respiration and temperature. I shall subsequently have occasion to show that paralysis of the vagi fully accounts for these. It is only necessary at present to state that the anomaly in the action of single doses of the drug, where the constitution is already affected by it, would be fully explained by the assumption that the vagi had ceased to be affected by its absorption, while the sympathetic was still stimulated by it.

Analysis of the physiological action.—What are the characteristics of the action of hyoscyamine when regarded

from a physiological point of view?   Harley, after numerous observations with a sulphate of hyoscyamine prepared by himself, came to the following conclusions regarding its action as compared with that of belladonna :—' The general action of henbane on the secretions and nervous system agrees in all respects with that of belladonna, and the results of its action are the same.   The difference between the two drugs may be summed up in these few words.   Compared with belladonna the influence of henbane on the cerebrum and motor centres is greater, while its stimulant action on the sympathetic is less.'[1]   With regard to its action on the heart and bloodvessels when given in moderate doses, Harley concludes that it ' affects the healthily excited heart in exactly the same way as belladonna influences the organ when unduly excited in the morbid processes.   Both drugs directly stimulate the heart, but after moderate doses the action of henbane results in a sedative effect.   Small doses of belladonna excite the heart, and large doses depress it.   Small doses of henbane are sedative and tonic to the heart, large doses excite it.   Excessive doses depress it almost as readily as those of belladonna.'   Dr. Harley believes that henbane is wholly removed by the kidneys, and founds his belief on the fact that the urine secreted during the action of the medicine dilates the pupil as readily as the same bulk of water to which the dose has been added.   I shall subsequently have occasion, however, to refer to observations which show that the excretory contents of the bowel have also the power of dilating the pupil, proving that a certain quantity of the drug is excreted through the intestinal canal.   By Dr. Harley it has been noticed in the cases which he examined that the diuretic action was, as a rule, more marked after the use of henbane than during the administration of belladonna, owing to the large increase of water.   By analysis also he determined that the urine contained an increase of urea, sulphates and phosphates, corresponding to that which is secreted during the action of belladonna.[2]   As Dr. Harley's conclusions regarding the action of hyoscyamus are expressed in terms of relation to the actions of belladonna,

[1] Harley, ' On the Old Vegetable Neurotics,' p. 337.
[2] *Loc. op. cit.*, p. 337–338.

it must be kept in mind that his explanation of the physiological action of atropia differs in some important particulars from that given by other observers. Thus, with regard to the action on the vagus, Dr. Harley controverts the opinion expressed by Meuriot that belladonna (and presumably hyoscyamus) accelerates the action of the heart by paralysis of the peripheral branches of the pneumogastrics, and that small doses have a quickening, and large doses a depressing effect on the respirations by paralysing the extremities of the same nerves. Meuriot founded his opinion on the fact that in animals under the influence of atropia, section of the vagi is not followed by any increase of the heart's action, and galvanisation of the nerve does not alter the movements of the organ. This observation has been confirmed by Bezold, Bloebaum, Wood, Lemattre, and others. As under ordinary conditions section of the *par vagum* greatly accelerates the action of the heart, and whereas when an animal is under the influence of atropia section produces no increase in the number of pulsations, these observers conclude that the greatly increased pulsation following either immediately or shortly after the administration of a large dose of belladonna, is due to special paralysis of the pneumogastrics. Dr. Harley does not apparently dispute the accuracy of the observation, but declines to agree with the deduction drawn from it. Commenting upon the effects of section of the pneumogastric under ordinary circumstances, he says :—' We are all familiar with the consequences. It is not that the heart is cut off from the influence of inhibitory nerves, as M. Sée, MM. E. and M. Cyon, and others would have us believe, but the organ is naturally excited to the utmost, and the pressure within the left ventricle is greatly increased by the engorgement of the pulmonary circulation. The heart is exercised to the full extent of its power, and neither belladonna nor any other agent can urge it beyond.' He believes ' that belladonna has no paralysing action on the vagus nerve, as exhibited in either the respiratory movements or in the action of the heart.' The quickening of the pulse produced by section of the vagus is, he affirms, precisely the same as the effect of belladonna in disease. ' When the pulse is at its maximum in fever, and when the lungs are perfectly free, the subcu-

taneous injection of atropia in either small or large doses
fails to increase it, and generally causes a diminution of its
frequency.' In this manner Harley appears to repudiate
the deduction, that because an animal under the influence of
atropia has its heart working at so high a pitch that section
of the pneumogastrics cannot accelerate its movements, the
condition must be dependent on paralysis of the vagi, inas-
much as the same negative result may be procured by the
administration of the drug under circumstances where
paralysis of the pneumogastrics is not at all suspected. By
other observers also it has been admitted, that though in
their opinion paralysis of the vagi is one of the principal
causes of the quickening of the heart's action in atropia
poisoning, it certainly is not the only one.

Lemattre has shown, and Wood has confirmed the ob-
servation, that even after section of the pneumogastrics the
action of the heart of an atropinised animal is quickened.
In my experiments the same result was procured with regard
to hyoscyamine. Bezold and Bloebaum have also demon-
strated that in animals under atropia the action of the heart
can be hastened by galvanisation of the thoracic sympathetic.
The conclusion arrived at by such observers is, that atropia
not only paralyses the pneumogastrics but stimulates the
sympathetic, and the generalisation most accordant with
all practical observations of the action of atropia and
hyoscyamine is, that the changes which occur in the thoracic
systems after the administration of sufficient doses, are
directly due to simultaneous stimulation of the sympathetic
system, and partial or complete paralysis of the cardiac and
pulmonary branches of the vagi. That hyoscyamine acts
specially upon the vagi has been to some extent shown when
the action of prolonged administration was referred to (p. 57),
and will again be incidentally commented on ; but direct
evidence of such an action can be obtained by certain special
experiments involving the section and medication of the
pneumogastrics themselves. When a rabbit is placed under
the influence of hyoscyamine, the pulse first becomes some-
what slower and the arterial pressure is increased. Subse-
quently the pulsations rise with almost certain persistency,
till the full action of the medicine is developed. If, shortly

after the drug has exercised its fullest effect, and is beginning
to be excreted and to lose its power, the pneumogastrics be
cut, it will be found that the pulse, which had begun to fall
towards the initial index, again returns almost precisely to
the highest point which it had reached during the operation
of the medicine. Thus :—

To a rabbit weighing 2lbs. 12ozs., one grain of the amorphous
extract of hyoscyamine was given. Before the administration the animal
was somewhat excited—Pulsations 160, respirations 140, temperature
101·8°, pupils 11½ points. The drug was injected hypodermically at 11.30.
At 11.40 the pupils were dilated to 13 points, the animal was weaker, but
could walk about fairly well. The fore-legs slipped from under it. It ex-
perienced great difficulty in turning round. It was excited and jumped
about without any apparent object. There was great tremor when it was
touched—Pulsations 180, respirations 155, pupils 13 points, temperature
102·1°. At 11.45 the pulsations were 170, the respirations 140, the pupils
14 points, the temperature 100·2°. At 12.10 there was great restlessness
with considerable loss of power. It lay with its chin flat upon the table,
and was drowsy. There was little or no loss of sensation—Pulsations 170,
respirations 118, temperature 102°. At 12.25 the animal was less restless,
and slept with intermissions. When the tail was pinched it struggled hard
to rise, but was unable to do so. When the irritation was desisted from
perfect rest ensued, the rabbit subsided at once, and lay flat and helpless.
Dry blotting-paper when placed in the mouth was only very slightly
moistened. There was impairment of sensibility about the face and
hind-legs, but the back and hips were much more sensitive, and showed
a greater capacity of reflex action. At 12.45 the pulse was 198, the respi-
rations 98, and the temperature 99·6°. The animal was now very prostrate
and lay perfectly quiet during examination, whereas formerly it struggled
severely. There was reflex closure of the eyelids, but no movement when
the inner surface of the ears or the face was irritated. The respirations
were now very abdominal. At 1.0 the pulse showed the first tendency to
decline, after having reached a maximum of 198 and counted 188 beats; the
respirations were 110, and the temperature 99·2°. The pupils still
measured 14 points on the pupilometer. At 1.10 the pneumogastrics
were severed. Immediately afterwards the pulse was counted, and was
found to measure 194, or only four beats below the maximum attained
during the action of hyoscyamine. Shortly afterwards, however, it began
and continued to fall, owing no doubt to an over-stimulation or exhaustion
of the sympathetic system, similar to that referred to by Dr. Harley in his
observations on the actions of atropia. In this instance it will be seen that
the temperature, after a slight rise, fell persistently till a loss of 4·1° was
attained. The pulse, as far as was determined, rose persistently from the
beginning, but no observation was taken during the first ten minutes, when
as a rule the initiatory depression occurs. The quickening reached a
maximum an hour and a half after administration, and at that time had
gained 38 beats per minute. The respirations fell persistently. Immediately
after section of the pneumogastric the pulse rose very nearly to the

maximum of its speed, and again fell gradually till death. Section of the pneumogastrics is not followed by such decided changes in rabbits as in carnivorous animals, so that in this experiment the rise was not decided; but in the following table, recording a similar observation on a cat, the quickening of pulsation was much more definite :—

TABLE IV.

Showing the action produced by section of the pneumogastrics in the Neck during the excitement produced by Hyoscyamine.

Time	Pulsations	Temperature
3·45	148	101°
(1½ grains)	—	—
4·5	163	100·5
4·30	155	100·5
4·45	255	100·4
5	240	100
5·15	200	99
5·25	195	97·9
(Pneumogastrics cut.)	—	—
5·35	240	97

In this observation 1½ grains were given to a cat at 3.45 P.M. For three-quarters of an hour there was a slight fluctuation of the pulse, then a sudden and maximum rise of a hundred beats, accompanied by the manifestation of great excitement, and followed by complete prostration. In the excitement the animal made snatches apparently at imaginary objects, and assumed unnatural attitudes. There were drowsiness and impairment of sensibility. The sense of hearing was, however, sufficiently acute to lead to the recognition of whistling and vocal sonnds. At 5.0 the mouth was absolutely dry, and the cat itself quite prostrate. When the tail was pressed upon the animal gave signs of pain, but was unable to move. There were occasional voluntary movements of the head, tail, and legs. The pulse had again fallen 16 beats, and the temperature continued to descend. From this time the pulse fell continuously. When it had reached 196, it was determined to ascertain what effect section of the pneumogastrics would have in again bringing it up towards the maximum point. At 5.30 the vagi were cut, and one minute afterwards it was found that the pulse had suddenly risen 44 beats per minute, and had almost reached the maximum attained 45 minutes prior to the section, and one hour after the administration of the medicine. The rise following upon the section of the inhibitory nerves shows either that the paralysis of the vagi had begun to pass off subsequently to the attainment of the maximum pulse, or that the sympathetic was irritated into more powerful excitement by the complete removal of the restraining power of the pneumogastrics. That the pulse did not reach to the full index characterising the maximum activity is again evidence that the sympathetic had been over-stimulated, and was not able to force up the heart to so rapid an action as it had attained an hour after the injection of the hyoscyamine. Previous to the section of the pneumo-

gastrics there was evidence that the paralysis of the vagi was being gradually removed, so that in this experiment it appears probable that the rapid and continuous fall of pulse after a rise goes to prove that the vagi which had been paralysed were undergoing progress towards the restoration of activity, while the inability of the accelerator nerves to raise the action to the maximum point, affords evidence that the sympathetic manifested after over-stimulation a weakness which was simultaneous with the returning power of the pneumogastrics.

The belief that the pneumogastrics are affected during the operation of hyoscyamine is supported by the observation that, if the vagi of a rabbit are exposed, isolated, and poisoned with hyoscyamine, very definite results follow. The symptoms relating to the respiration and cardiac action are exactly the same as during the constitutional action of the drug. The temperature also is characteristically reduced, but no motor symptoms are developed. I exposed the pneumogastric nerves of a rabbit, and smeared them after isolation with hyoscyamine. Immediately the pulse rose from 162 to 270 beats, the respirations were reduced one-half, and *in forty-five minutes the temperature had fallen* 3°. The pulsations remained high, and the temperature and respirations low, for two hours, and in three hours had almost regained the starting-point. I think the marked reduction of temperature is sufficient to show that the cardiac excitement was not the result of the operation performed, and the accordance of the symptoms with those in cases where hyoscyamine has been injected hypodermically, seems to me to indicate that whatever may be the action of atropia there is no doubt that one of the actions of hyoscyamine is to paralyse the pneumogastrics. The relation between the decline of temperature and the character of the respirations produced on section ofthe vagi, affords, as will be subsequently shown, additional evidence that this deduction is a reasonable one.

But besides the action on the sympathetic and the vagi, the foregoing experiments show that there is a decided physiological influence exerted on other organs and tissues. The most important of these are primary stimulation of the corpora striata and of the hemispheres, followed by marked irregularity in the function of the latter, and a

peripheral suppression of the activity of the former. This primary stimulation is followed by delirium with motor excitement, and subsequently by muscular prostration resulting from the inability of the palsied motor nerves to transmit central impressions.

Special actions on systems and tissues.—One of the earliest and most persistent appearances produced by hyoscyamine is dilatation of the pupil, and the aberrations of vision and paralysis of accommodation following upon that dilatation or caused by contraction of the ciliary muscle. These conditions are exactly analogous to the intra-ocular changes produced by atropia, and are consequently amenable to the same explanations. There can be little doubt, in the present state of our knowledge of physiology, that the circular fibres of the iris under the control of the third pair are those the action of which causes contraction of the pupil, while the radiating fibres supplied by the cervical portion of the sympathetic act so as to produce dilatation. The trigeminus also has some influence, reflected through encephalic centres,[1] on the operation of the radiating fibres. It has been a matter of extensive investigation and wide diversity of opinion how the mydriatic substances act in producing dilatation of the pupil and the associated ocular changes. In accordance with his ideas of the action of atropia, Harley believes that these ocular changes are induced by the stimulation of the sympathetic, which simply 'overpowers the action of the third nerve. The sphincter gradually yields to the stronger contractions of the radiating fibres, and the pupil opens. As this occurs the action of the circular fibres is progressively weakened, and when the sphincter is fairly overpowered it no longer affords an unyielding line to the contracting fibres, and these having now no fixed support for *inward and convergent* action, are thus rendered incapable of compressing the lens.[2] Harley has made an interesting observation, which in his opinion appears to militate against the opinion that the third nerve is paralysed by the mydriatics. On bringing the subject

[1] Dr. C. Radclyffe Hall, 'Edin. Med. and Surg. Journal,' 1846–48, quoted by Carpenter.

[2] *Loc. op. cit.* p. 237.

of experiment from the shade into a strong light, after the injection of atropia he found that the pupils were more contracted than under the same external condition before injection. He concluded that the third pair had been roused to unusual exertion just at the time when the increasing influence of the sympathetic began to be first felt, and that the sudden stimulus of light had called forth its opposing energy to such a degree, that for a few minutes it was able to repress the rising force of the sympathetic, which a little later on would become overpowering.[1] Dr. Harley's opinions regarding the influence of dilatation of the pupil in causing alteration in the form of the lens is liable to modification by the case of Graefe, where the iris was wholly removed by operation and the power of accommodation remained.[2] The conclusion derivable from this case is that the change in convexity of the lens is produced by the pressure occasioned by contraction of the ciliary muscle. Dr. Wood[3] enters very fully into the discussion of this question, and furnishes a very comprehensive review of the various experimental researches, and the deductions derived from them, by English, Continental, and American observers. In support of the opinion that dilatation of the pupil by local application is independent of nerve centres farther back than the ciliary ganglion, he states that he has seen atropia-mydriasis occur in cases of complete oculo-motor paralysis in man, just as Claude Bernard and Lemattre demonstrated it in animals after section of the third pair. He also accepts the testimony of numerous observers, and the results of his own experiments and clinical observation, as showing that dilatation occurs after section of the trigeminus or of the cervical sympathetic, or both. Dr. Wood's intention in advancing these doctrines is to lead to the proof of his opinion that in the case of atropia, at least, the dilatation of the pupil by local application is produced by an action of the medicine on the nerve endings in the iris. For the

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[1] Harley, *op. loc. cit.* pp. 231–32. Is it not possible, however, that this contraction was due to irritation of the third nerve preceding paralysis?

[2] ' Archiv f. Ophthalmologie,' B. vii.

[3] *Op. loc. cit.* pp. 218–222.

purpose of doing so he quotes the experiments of Bernstein, Dogiel, and Engelhardt as proof positive that dilatation by local application of atropia can be produced independently of the ciliary ganglion, and refers to numerous observations, in which the pupils of extirpated eyeballs and the eyeballs of the dead have been dilated by mydriatics. It appears to me, however, that the last observation as recorded by Borelli, in the 'Edinburgh Medical Journal,' for November, 1871, proves too much for the interests of Dr. Wood's cause, inasmuch as in the eyes of the dead the nerve endings could only have the same relation to the muscular tissue of the iris as the ciliary ganglion had to it when that body was, in the living, cut off from communication with it. It follows that if the production of artificial dilatation of the pupil after removal of the ciliary ganglion affords proof that the movements of the iris are independent of that centre, the similar production of it after death also proves that the dilatation is independent of the operation of the nerve endings themselves. This conclusion can only be avoided by the assumption that the nerve endings are independent centres capable of producing reflex action, or that the muscular structure of the iris is directly acted upon by the drug—both of which suppositions Dr. Wood repudiates.

Another question on which opinions vary is, do mydriatics act by stimulating the sympathetic, by paralysing the third pair, or by both ways? As I have already had occasion to state, Dr. Harley ascribes the action entirely to the sympathetic, repudiates the assertion of Meuriot that atropia paralyses the oculo-motors, and contrasts the action of that drug with the intra-ocular action of conium, which undoubtedly does paralyse the ciliary branches of the third pair. He also advances the fact that contraction of the pupil occurs during sleep as proof that the third pair is not paralysed in atropia-mydriasis. Donders and other eminent observers, however, share Meuriot's opinion as to the existence of impaired activity in the oculo-motor nerves, and the numerical balance of opinion inclines to the belief that a decidedly depressant action on the third pair constitutes part of the process by which the pupil

dilates after the local or constitutional use of atropia and other mydriatics of the same class. Is there also a stimulating action of the sympathetic? The well-known observations that the dilatation produced by atropia is sufficiently active to lead to the rupture of adhesions, and that the pupil in paralysis of the oculo-motor nerve is not nearly so much dilated as it can be by the use of atropia, constitute sufficient proof that the sympathetic is affected.

Wood also endeavours to establish that, when atropia is administered internally, the action on the pupil is, as in local applications, produced by the influence of the drug on· the peripheral endings of the iridial nerves. The observations of Lemattre that when the humours from the eye of a dog, or even from the fœtus of an animal poisoned by atropia, are applied to a normal eye, dilatation of the pupil results, constitute part of the evidence on which Dr. Wood bases this opinion. Donders and himself, however, have not succeeded in producing the same result, and an observation to which I have already referred did not, as far as the analogous drug hyoscyamine is concerned, coincide with the last part of Lemattre's statement. I poisoned a pregnant cat with hyoscyamine, and as one of the kittens came to life immediately before the death of the animal, I cut down upon it two or three minutes after breathing had stopped, and by artificial respiration kept the kitten alive. It lived nearly four hours, breathing and moving naturally, and did not present a single sign of having been at all affected by the drug. Neither did the liquor-amnii produce dilatation in another animal's pupils. The mere fact also that the contents of the eyeball contain a sufficient quantity of a mydriatic drug to cause dilatation in a normal eye, is no proof whatever that the drug acts directly on the peripheral ends of the nerves of the iris. A clinical case is advanced by Dr. Wood, in which there was complete paralysis of the facial, the trigeminus, and the oculo-motor nerve, and in which ' the carotid canal was so pressed upon, that the sympathetic nerve which passed upwards through it to the eye must also

have been paralysed.' In this case, atropia injected hypoder-
mically caused dilatation of the pupil, and the author
contends that the action must have been peripheral. The
evidence would be more complete if the existence of absolute
paralysis of the sympathetic distribution to the eye were
more certainly established, and the only conclusion which
the evidence bearing on the whole subject justifies, is that
artificial dilatation of the pupil results from stimulation of
the sympathetic, and partial paralysis of those fibres of the
oculo-motor nerve which regulate the contraction of the
pupil.

I have entered somewhat minutely into this question,
inasmuch as the action of hyoscyamine on the pupil is
exactly analogous in character, and most probably in cause,
to that resulting from the use of atropia. That action
has been already illustrated and discussed in this paper.
In recapitulation it is sufficient to state that by local
application of hyoscyamine in solution the pupil is fully
dilated in a few minutes, and that the dilatation lasts for
several days. The urine of animals constitutionally affected
with the drug causes a dilatation equal to that produced by
the solution. I have several times demonstrated that the
alvine excreta of animals poisoned by hyoscyamine also
produce dilatation in normal eyes. The most decided ob-
servation of this sort consisted in smearing the eye of a rabbit,
whose pupil measured twelve points on the pupilometer,
with fæcal matter from the colon of a cat. The dilatation
was slow, but by next morning the pupil had reached a
diameter of sixteen points. The full dilatation was persistent
during the day, and the diminution was a very gradual one.
Perhaps more than the usual amount of the drug, however,
was present in the fæces of this cat, as there was marked
retention of urine. The liquor-amnii from the same cat
did not produce dilatation. A strong solution of Calabar
bean, containing an amount of extract of physostigma,
equal to four grains of powder to the minim of water, did
not counteract or prevent the dilatation produced in animals
by hyoscyamine, either when the latter was applied locally
or when it was given hypodermically; neither did this strong

solution of Calabar bean produce contraction of the human pupil after accidental or intentional dilatation by hyoscyamine.

When the drug is administered internally, the dilatation is more lasting than when it is applied locally. Thus the pupils of a rabbit, which had not been previously employed for observation, were found to measure $11\frac{1}{2}$ points on the pupilometer, each point being one-fortieth of an inch. Two drops of a solution containing 1 grain in 10 were placed in the eye. In four minutes the pupil measured $12\frac{1}{2}$ points, in ten minutes 14, and in fifteen minutes 15 points. Three and a half hours after the application, the pupil was again reduced to 14 points. For the next two days it measured $13\frac{1}{2}$ points, and on the third day was contracted to the initial diameter of $11\frac{1}{2}$ points. But where 2 grains were hypodermically injected into another rabbit, the pupils were in two minutes dilated from $11\frac{1}{2}$ points to 14. In a quarter of an hour they measured 15, and in five minutes more had reached the condition of fullest dilatation, namely, 16 points. This measurement remained persistent till next day, when there was a diminution of half a point, followed by a gradual contraction, which did not reach the starting-point till the sixth day after administration. It appears, therefore, that after the constitutional use of the drug dilatation is much more rapidly produced, and considerably more persistent, than when induced by local application. A corresponding duration characterises the dilatation of the human pupil, according to the method of its production.

Action on the secretions.—The most noticeable early action of hyoscyamine is the production of great dryness of the mouth and throat, and subsequently of the lips, by diminution or modification of the salivary and buccal secretions. This dryness, as I have frequently seen, is not produced in pigeons, and it is not improbable that this fact may be the means of leading to some explanation of the anomalous action of the mydriatics when administered to birds. It is possible that the non-dilatation of the pupil and the unimpaired production of buccal and salivary secretion may be traceable to a variation in the distribution, or a modification in the function of

the trigeminus of birds. In rabbits and guinea-pigs hyoscyamine never causes sickness, either when given by the stomach or when administered hypodermically. In cats and dogs, however, sickness almost invariably follows upon its hypodermic use. Single doses of hyoscyamine appear to produce constipation, evidently as a result of diminution of intestinal moisture. Fæcal matter is seldom passed during the operation of a moderate dose, and when excreted at all it is exceedingly scanty, hard and dry. The alvine excretions are proved to contain hyoscyamine by the application of them to a sound eye, producing slow but certain dilatation. The urine, especially after moderate doses, is increased in quantity and is forcibly expelled. It contains the great bulk of the drug, and when applied to a sound eye produces dilatation as readily as a similar quantity of the solution employed. In larger doses, however, hyoscamine sometimes leads to retention, but more commonly to suppression of urine, followed by coma and death.

Action on the circulation.—When very small doses of hyoscyamine are administered, the effect, if the animal has been excited, is to reduce the pulse numerically to such a condition as is characteristic of repose. At the same time the arterial pressure is increased, and the cardiac and femoral pulsations become more readily perceptible than before. If sufficient of the drug is given to cause excitement, the pulse is slightly and temporarily elevated simultaneously with the production of dryness of the mouth and throat. When somewhat larger doses are administered, there is first a gradual slowing of the pulse, which may continue during the first half-hour, but much more commonly lasts only for about ten minutes. Then, synchronous with the manifestation of a tendency to sleep between paroxysms of excitement, the pulse rises with a sudden bound. At the same time the dryness of the fauces is intense, and the animal shows defective accommodation of the eyes to near objects. The pulse continues quick and feeble for about half an hour, and when the developed symptoms are accompanied by a free secretion of urine, it sinks almost to the starting-point with the same suddenness which characterised its ascent. After still larger

doses there is little or no preliminary depression of the pulse, but an immediate increase both in the number and force of the pulsations. Excitement and motor symptoms are at the same time developed. This great rapidity of the pulse, which is accompanied by slowness of the respirations and diminution of temperature, continues throughout the whole of the somnambulistic stage. It is suddenly modified on the return of motor power, when the animal unexpectedly wakens up and walks about with comparative agility. After fatal doses there is an immediate and considerable rise of the pulse, with increase of arterial pressure ; then as prostration follows upon the stage of excitement, the pulsations become suddenly inappreciable, and remain so till the approach of death. During this period the animal usually sleeps heavily, but with intermissions, which originate in subjective conditions. Towards death the pulse is again felt beating in a slow and laboured manner. At the same time the temperature and the respirations suddenly fall, the pulse remains sluggish till the fatal issue, and, in some instances, after breathing has been stopped for 75 minutes, the auricles and sometimes the ventricles may be seen on exposure making rhythmical movements, which become slower and slower till complete cessation is reached. The administration of rapidly fatal doses causes an excessive and sudden rise in the rapidity of the pulse, and leads to death by syncope during the maximum activity of the heart, the beatings of which may increase from 140 to 285 in the course of an hour. In such cases the heart's action generally fails in a paroxysm of delirious excitement. Thus it will be seen that small doses of hyoscyamine have a tonic action on the heart and vessels through stimulation of the sympathetic ; that large doses primarily depress and subsequently excite the heart's action ; that still larger doses cause direct vascular irritation ; that poisonous doses induce great primary excitement, followed by marked and continuous cardiac and respiratory depression ; and that still larger lethal doses lead to sudden syncope during the height of cardiac excitement. It has been already seen that hyoscyamine does not enter the placental circulation, as the kitten of a cat poisoned by

the drug lived for four hours without presenting a single symptom of henbane poisoning.

Action on respiration.—The effect of small doses of hyoscyamine on the respiratory movements is similar to that upon pulsation. It brings them to a condition significant of repose. Under the influence of larger doses, however, there is a great decrease in the number of the respirations simultaneous with the rise in pulsation, and fluctuating with the modifications of the pulse. In still larger doses, such as that administered to the dog (Table No. II.), the character of the respirations, and their numerical ratio are exactly the same as are produced by section of the vagi. The respirations become deeper than before, and decidedly diaphragmatic. The abdominal muscles are markedly active in inspiration. The inspirations are slow, long, and subdivided into three or four separate acts, which are sometimes hiccupy in character; the expiration is abrupt, and followed by a long pause. The respirations are diminished in number one-half. Thus, in a dog, they were reduced from 36 to 18, after having risen to 78 in the stage of excitement. When poisonous doses are given, the respirations sink in the ratio of from 36 to 13 per minute, and have exactly the same character as those just described. When in such cases the body is opened shortly after death, the diaphragm is found making five rhythmical contractions every minute. The character of the respiration in animals under the influence of large doses of hyoscyamine appears to me to be much more indicative of the presence of paralysis of the pneumogastrics than the changes induced in the pulse, especially when taken in connection with the steady and invariable decline of temperature which always occurs during the existence of the abdominal and diaphragmatic respiration. In such conditions of the respiratory function section of the vagi only intensifies the length and jerkiness of the inspiration to a slight degree, and leads to a fatal result much more quickly than in animals not affected by hyoscyamine.

Action on the temperature.—Hyoscyamine induces in animals a reduction of temperature. After small doses the reduction is slight. Thus in a rabbit, to which half a grain of the amorphous extract was given, it fell only 1°. When,

however, double that dose was administered to a rabbit of nearly the same weight, the fall was 2·6°, accompanied by an increase of 50 pulse-beats and a diminution of 42 in the respirations. Three grains administered to a cat brought down the temperature 12°, while it raised the pulse 40 beats, and reduced the respirations from 42 to 18 per minute. When the pulse again fell and the respirations rose, the temperature was steadily elevated to the starting-point. In a dog, similarly treated, the fall was 2·8°. In another cat, poisoned with 5 grains, the fall of temperature up to the time when the animal became moribund was 8·2°. These observations regarding the great fall in temperature, associated with quickening of the pulse and diminution of the respirations, confirm the opinion that the *par vagum* suffers in poisoning by hyoscyamine. The condition induced by section of the vagi illustrates this statement. 'Although section of both vagi does not materially either increase or diminish the work done in a given time by the respiratory muscles, it interferes very considerably with the accomplishment of the purpose of their movements—the arterialisation of the blood.' [1] In all cases of *post mortem* examination after poisoning by hyoscyamine, the blood has been found dark and fluid, a condition which also exists in death after section of the pneumogastrics. Preceding this affection of the *par vagum*, however, there is no doubt a marked stimulation of the sympathetic system, as evidenced by the changes in the heart and circulation which have been already referred to.

Action on the cerebral centres.—In order to determine the vascular changes, in the cerebrum, I exposed under chloroform one hemisphere of a rabbit and observed the condition of the intra-cranial circulation. Time was allowed for the production of changes incidental to the exposure to atmospheric irritation and the removal of vascular support. A sufficient dose of hyoscyamine was then given, and almost immediately there was a marked contraction of the vessels under observation. As the pulse quickened there was slight dilatation, but subsequently little or no change occurred till shortly before death, which took place in about an hour. Previous to death the number of visible branches of the main trunk which had been selected,

[1] 'Handbook of the Physiological Laboratory.' Burdon Sanderson. P. 808.

was reduced from 13 to 10, and after death the other side of the brain was found pale and anæmic. This condition of the brain was existent in every case where there had been no retention of urine. It was observed, however, that the *corpora striata* were more vascular than any other part of the brain substance. These appearances, and the symptoms already recorded, lead to the inference that the first effect of hyoscyamine is stimulation of the *corpora striata* and cerebral hemispheres, which at first gives rise to symptoms of excitement and constant muscular movement, conditions which are subsequently counteracted or subdued by the presence of paralysis of the motor nerves. When large doses are given, when respiration becomes greatly impeded, or especially when there is marked retention of urine, the brain suffers from secondary irritation, due to aberrations from the normal qualitative and quantitative standard of the blood; and delusions, incoherence, and other symptoms of cerebral disorder, ensue.

Action on the motor nerves.—That the motor nerves are themselves paralysed by hyoscyamine, is proved by the fact that, when the femoral artery is tied previous to the administration of hyoscyamine, the limb thus protected retains motor power much longer than the opposite one. This experiment, which was employed by Botkin in investigations regarding the action of atropia, I have repeated, and have found that in the earlier stages of the action of the medicine a considerably greater amount of motility is shown by the leg of which the main artery is occluded than can be elicited by simple or electrical stimulus from the corresponding limb on the opposite side.

Action on the sensory nerves.—In the latter stages of the action of hyoscyamine there is decided impairment of general sensation. This can be tested during intercurrent periods of excitement in the somnambulistic stage. The nerves of special sense are little, if at all, affected, as animals labouring under the influence of hyoscyamine can hear distant shrill whistling, and repeatedly struggle to reach or avoid visible objects till they sink into the moribund condition.

Having completed the record of these investigations regarding the actions of hyoscyamine when administered to the lower animals, it is necessary to inquire into the effects

produced on the human species by the physiological employment of the drug.

Action on man.—The conditions which result from the administration of hyoscyamine to man are similar to those which are produced in animals, and in equal quantities the drug is almost as powerful when given to the former by the mouth, as when injected hypodermically into the latter. Two grains of the solution were given to an adult at 11.50 A.M., three hours after a meal. Previous to administration the pulse was 104; the temperature in the arm-pit 98·6°, and that in the mouth 98·9°; the respirations were 20 per minute, and the pupils $\frac{1}{8}$th of an inch in diameter. The following table will show the alterations produced in the system by a two grain dose of the amorphous extract, ten minutes after the administration, and during the course of the action of the drug.

TABLE V.

Physiological action on Man of two grains of Hyoscyamine.

Time	Pulse	Resp.	T. Mouth	T. Axilla	Pupils	Remarks
			Degrees	Degrees	Inch	
11·30	104	20	98·9	98·6	$\frac{1}{8}$	
11·50	—	—	—	—	—	Two grains of hyoscyamine.
12	94	18	98·9	98·6	$\frac{3}{8}$	
12·10	80	18	98·6	98·6	$\frac{3}{16}$	Mouth dry. Giddiness.
12·20	116	20	98·7	98·7	$\frac{1}{4}$	Motion impaired.
12·30	116	20	98·9	98·7	$\frac{1}{4}$	Drowsiness.
12·40	120	24	98·9	98·5	$\frac{1}{4}$	Motion more impaired.
12·50	120	24	98·9	98·9	$\frac{1}{4}$	Articulation slow.
1	114	22	98·8	98·7	$\frac{1}{4}$	Mouth and nose very dry.
1·10	108	24	98·5	98·5	$\frac{1}{4}$	Interrupted sleep.
1·20	105	22	98·2	98·2	$\frac{1}{4}$	{ Illusions and paralysis of ac-
1·30	100	21	98·2	98·2	$\frac{1}{4}$	{ commodation.
1·40	105	22	98·5	98·2	$\frac{1}{4}$	{ Active dreams and involuntary { exclamations.
1·50	106	20	98·9	98·6	$\frac{1}{4}$	Icterus.
2	100	21	98·2	98·2	$\frac{1}{4}$	Movement still impaired.
2·10	94	18	98·3	98·2	$\frac{3}{8}$	Incoherence and aphasia.

It will be seen that during the time embraced in this table there was a fall of temperature, which was at its lowest when the development of delirium rendered it impossible in this particular instance to take accurate observations. It appears also that this decline was not continuous, but was broken by a temporary and slight rise just at the

commencement of the manifestation of the physiological action. At this time also the temperature in the mouth was relatively low, being equal to that in the axilla, which was at other times three or four decimal points under it. Another rise occurred just at the beginning of the mental excitement, but the variations of temperature only passed through a space of seven decimal points in all. The pulse showed corresponding variations, being first slackened, then accelerated ; then, as sleep was almost overpowering, it again became temporarily reduced. Almost simultaneously with the appearance of drowsiness, there occurred a slight attack of icterus, due apparently to non-elimination of bile.[1] There was no diuretic action, either at the time of or after the generally recognised symptoms produced by the drug. After the pulse, &c., were no longer recorded, the mental state was noticeably characterised by heaviness, incoherence in conversation, and a certain amount of ataxic aphasia, as shown by a tendency to slur over words and syllables. During the following night sleep was disturbed by dreams, in which the person affected frequently made short but audible remarks, evidently in answer to subjective interrogations.

In order to test the action of a larger dose, three grains of the amorphous alkaloid were, after an interval of several days, given to the same patient. The following table will illustrate the action produced.

TABLE VI.
Physiological action on Man of 3 grains of Hyoscyamine.

Time	Pulse	Resp.	T. Mouth	T. Axilla	Pupils	Remarks
			Degrees	Degrees	Inch	
3·50	104	20	99·5	99	$\frac{3}{16}$	Three grains given.
4	108	19	99·2	99·1	$\frac{3}{16}$	Lips dry. Giddy.
4·15	112	22	99·2	99	$\frac{3}{16}$	Great loss of motor power.
4·30	108	19	99·2	99	$\frac{1}{4}$	Marked drowsiness.
4·40	116	23	98·8	98·7	$\frac{1}{4}$	Voice husky. Interrupted sleep.
5·10	126	22	99	99	$\frac{1}{4}$	Could not walk alone.
5·23	110	22	99	99	$\frac{1}{4}$	Interrupted sleep.
5·30	114	—	—	98·9	$\frac{1}{4}$	Restless and incoherent.

[1] The subject of the experiment was the only person who witnessed the existence of jaundice. As it has never been seen in the course of numerous subsequent experiments, it would be premature to say that the appearance was not due to ocular and cerebral changes in the subject of the observations.

For seven hours subsequent to the last period recorded in this table, it was possible to take only occasional and irregular observations. During a long-continued display of delirious excitement the pulse fluctuated irregularly between 104 and 120, the respirations fell to 14, and the temperature reached a minimum of 98·4°, and a maximum of 99·2° in the axilla. During this stage of the action of the medicine the patient manifested all the symptoms of simple mania. He mistook identities, spoke incoherently, acted irrationally, and was frequently under the influence of delusions and hallucinations. Early in this period there was marked paralysis of ocular accommodation. Chairs, which were five or six feet distant, were grasped at as if within reach, and repeated efforts were required to rectify the mistakes originating in this aberration of vision. The full cerebral effect of the drug was manifested two hours after administration. The first symptom was the inability to fix the attention on any given subject. The patient, though rising from his seat for a definite purpose, immediately forgot what his own intentions were. He talked in incoherent snatches, and after commencing to express an idea, passed suddenly on to the statement of another and apparently unconnected one, which also he left obscure and unexplained. While under the combined influence of delusions, hallucinations of sight, and paralysis of accommodation, he attempted to step from a window about thirty feet from the ground, stating that he was going out upon the lawn, which he was convinced was on a level with the window. He had numerous hallucinations of sight. A picture which, under the influence of a delusion, he had taken from the wall and placed on a sofa, he immediately afterwards sat upon in the belief that it was an embroidered cushion. He pointed to cats which, he said, were standing on their hind-legs, and making fantastic movements, when no such animals were visible to others. Friends at a distance were spoken to as if sitting in parts of the room which were quite unoccupied. Female heads and faces were pointed at in the air as examples of perfect grace and beauty. The mistakes in identity were so complete that interrupted conversation was carried on with persons who had temporarily assumed the identity ascribed to them by the patient. During the

persistence of these personal delusions, statements, which were either unreservedly sincere or ludicrously inappropriate, were freely elicited by suggestion. The patient several times partially changed his clothes with the intention of going out for special purposes—to work, to walk, or to dine—but every new suggestion led to a modification of dress till, actuated in a totally different manner, he left his toilet incomplete, and directed his attention to new and equally absurd pursuits.

Latterly the motor impairment became less marked, but the cerebral condition was characterised by greater incoherence in speech and extravagance in action. The patient tried repeatedly to wind up a gold watch with a corkscrew. Ideas were imperfectly expressed, which at best had neither sequence nor relative conuection. The patient walked in an aimless manner from room to room, but to a great extent avoided obstacles in his way. The emotional condition was one of quiet satisfaction and comparative good-humour. When the influence of delusions required the use of personal restraint, the patient met it more by illogical argument and expostulation than by force. The cerebral condition during the time following upon the short period of drowsiness was characterised by wakeful restlessness, till nine hours after the administration of the medicine the patient, in a comparatively lucid interval, undressed and went to bed. Throughout the night he experienced numerous hallucinations of sight. Lizards and other animals crawled on the bed-clothes. A face—always the same—formed itself by the hollows and elevations of the cornice, but this delusion could be dispelled by mental analysis. Two sides of the room appeared to form the two pages of a book separated by the intervening angle; and by the influence of hypermetropia, seemed to rest upon the bed, and lie open before the patient; but after fixed attention the vision appeared to recede, till the elements of the delusion were fully determined by their relation to surrounding objects. After a short period of interrupted morning sleep, no traces of the operation of the drug remained, except wide dilatation of the pupils, some dryness of the throat, and slight lassitude. During the action of the drug there was no

reddening of the skin, or other appearance of eruption.[1] Though during the greater part of the time consciousness was not affected, scarcely a single incident anterior to the time of going to bed was remembered by the patient, neither could the events recorded be recalled to his memory by any attempt to associate the vagaries which he had forgotten with those of which he had a dim recollection. While the physiological action of the drug lasted there was free diuresis, but no vomiting or nausea.

In this case, as in the former, it will be seen that the action of hyoscyamine on man is analogous to that on the lower animals. In a patient under the influence of a large dose, the pulse quickens and the temperature inclines to fall. In one important respect, however, there is a difference in the distribution of the physiological action. In man the respirations and the temperature are relatively very little affected; the motor paralysis is imperfect and transient, while, on the other hand, the affection of the cerebrum is decided and prolonged. In accordance with what I have already said regarding the causal relation between the alterations in respiration and temperature and the paralysis of the vagi, it appears to me probable that this modification in the symptoms produced in man, as compared with those recorded as occurring in the lower animals, is explicable on the ground that in man the pulmonary portion of the vagus is less affected than the cardiac, while in animals both parts are equally paralysed. When the therapeutic standing of the drug is established on a scientific basis, it will no doubt be found that this modification is a most beneficial one to man, as it may be the means of permitting the substance to exercise a potent influence on the cerebrum without involving the dangers which would follow upon retardation of the respirations or sudden reduction below the normal index of the temperature.

Summary.—For the better recollection of the isolated statements which have been brought forward in this paper,

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[1] Since the time that this paragraph was written, I have had frequent occasion to see and demonstrate a decided rash, produced by the use of hyoscyamine. It appears most frequently on the face and forearms, and is not unlike the eruption of measles.

it is necessary to summarise the conclusions which have been arrived at.   It has been seen that—

1. The smallest active doses of hyoscyamine when administered to animals cause numerical depression of the pulse and increased arterial tension, reduce the temperature, dilate the pupil, and have little effect on the respirations.

2. Small doses cause reduction of the pulse, with increase of arterial pressure, followed by quickening, which after a greater or less persistence somewhat suddenly subsides and sinks towards the normal point.   During the action of the medicine there is restlessness, followed by motor paralysis, diminution of respiration, and reduction of temperature about 4°.   The drug in all cases produces dilatation of the pupil and dryness of the mouth and throat.

3. Large doses elevate the pulse without previous depression, and this quickening is maintained for six or eight hours.   Great fall of temperature, diminution of the respirations, loss of motor power, delirious excitement, and sometimes prolonged but interrupted sleep, succeed, and are in their turn followed by sudden reduction of the pulse towards the initial point, and sometimes below it.   This fall precedes complete recovery from the operation of the drug.

4. Lethal doses cause death either by syncope during extreme vascular excitement, or by coma following upon nonelimination of the drug by the urine and imperfect æration of the blood through impairment of respiration.

5. The drug generally produces increased urinary and diminished alvine secretion.

6. To rabbits, pigeons, and others of the lower animals, hyoscyamine is almost, if not altogether, as active a poison as to man ; but in birds no dilatation of the pupil is produced by it, and no dryness of the mouth and throat.

7. Long-continued administration of the drug causes loss of weight, quickening of the pulsation with increase of arterial pressure, quickening also of respiration and *increase of temperature.*   Subsequently there is restoration of weight, but persistence of heightened pulsation, respiration, and temperature.   Individual doses administered to an animal constitutionally affected with hyoscyamine, cause not reduction, but elevation of animal heat.

8. The intra-thoracic changes and the decline of temperature are physiologically produced by stimulation of the sympathetic system and depression of the cardiac and pulmonary distribution of the pneumogastrics. The elevation of temperature caused by individual doses administered to animals persistently under the influence of the drug, is probably due to a tolerance of the substance by the pneumogastrics, while the sympathetic still remains affected by it.

9. The pupil is dilated by hyoscyamine through simultaneous stimulation of the sympathetic and depression of the third pair as distributed to the iris.

10. Local application causes dilatation of the pupil, commencing in from three to four minutes, increasing for fifteen minutes, persistent for about three and a half hours, gradually passing off and returning to the normal state in three days. After the internal use of moderate doses the pupil begins to dilate in from two to three minutes, and reaches the widest diameter in twenty minutes. The maximum dilatation persists more than twenty-four hours, and the pupil does not return to its normal size for six days.

11. In man the cerebral symptoms are more marked than in the lower animals, and the motor, cardiac, respiratory, and thermal symptoms less so. With small doses the pulse is first slightly reduced, and at the commencement of the cerebral and motor excitement is elevated about twenty beats above the initial index. The pupil is dilated and the vision becomes imperfect. During the hypnotic stage the pulse falls towards the starting point. During the whole period the temperature falls 0·7°, and after the cessation of interrupted sleep some cerebral confusion remains, as is manifested by occasional incoherence and a liability to trifling delusions.

12. Larger doses cause direct elevation of the pulse and slight reduction of oral temperature. Dilatation of the pupil is followed by paralysis of ocular accommodation. Motor power is impaired, and interrupted sleep alternates with, and is followed by, delirium. Delusions and hallucinations associated with rapid and imperfect ideation are succeeded in six or seven hours by a renewed tendency to sleep,

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which is disturbed by dreams and by intervals of wakeful-
ness with hallucinations. The symptoms during this action
on man are divisible into four stages. There are (1) slight
rise of the pulse simultaneously with the production of dila-
tation of the pupil, and giddiness and dryness of the mouth
occurring in about five minutes ; (2) drowsiness and impair-
ment of motor power, reaching a maximum in an hour and a
quarter ; (3) wakefulness, restlessness, incoherence, and the
production of delusions and hallucinations ; and (4) returning
drowsiness and sleep alternating with short periods of waking,
which are characterised by a repetition of a limited number
of hallucinations.

What suggestions as to the therapeutic value of hyos-
cyamine can be drawn from the review of the physiological
actions of the drug? Little can be said of it from this
point of view that has not already been said of hyoscyamus
itself, and even little can be asserted about this drug that is
not to a considerable extent based on the analogy that exists
between it and belladonna. The leading and most readily
produced actions of hyoscyamine are those which depend on
the stimulation of the sympathetic and vaso-motor systems.
In fact, it appears probable that small doses have no other
action whatever than to cause an increase of arterial pressure
and a numerical depression of the heart's action, associated
with greater force and regularity through the direct stimula-
tion of these systems. But the condition induced by such an
action on the blood-vessels is accompanied by secondary
changes, which themselves indicate conditions in which the
drug should be medicinally useful. Thus the marked diu-
resis which accompanies, if it does not depend upon, the in-
crease of arterial pressure, points to hyoscyamine as useful
in various forms of nephritis. The influence which the regu-
lation of the action of the heart has upon the removal of
spasmodic affections of the respiratory function, such as
asthma, suggests the use of hyoscyamine in the treatment of
such diseases. The gentle and uniform stimulation of the
sympathetic system must also afford an explanation of the
generally accepted belief that the crude drug prevents the
intestinal griping of purgatives, inasmuch as when given for

such purposes it is administered in doses which would be insufficient to act upon the sensory nerves. Such an action on the sympathetic would in all probability lead to an equability of peristalsis and avert the pain arising from abnormal contraction in one, and abnormal dilatation in another part of the bowel. The effect of somewhat larger doses of hyoscyamine in slightly raising the pulse in man, while still increasing the blood pressure, is evidence that the medicine is a reliable and general stimulant. It increases the force of the circulation, and provides for a more uniform and larger supply of blood to the tissues. The character which it has acquired as a means of removing neuralgic pains in the viscera, and allaying irritability of the genito-urinary system, shows, in addition to the evidence I have had occasion to advance regarding its action on the sensory nerves and its liberal excretion by the kidneys, that its employment in such cases is warranted by the results which at any time could be fairly anticipated and have already been widely experienced. There can be no doubt also that in many cases of maniacal excitement, hyoscyamus and its active principle can produce comparative, if not complete, quiescence.[1] This is practically most evident in the outbursts of excitement which accompany the progress of senile dementia. In many such cases the drug has an almost immediate effect, either in bringing the mind back to the condition of unobtrusive dementia, or in substituting for the noisy excitement of senile mania the comparative calm and quietude which characterise the cerebral action even of large doses of henbane. In mania, and in epileptic excitement, in the delirium of fevers, in meningitis, delirium tremens, and all forms of direct or secondary cerebral

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[1] Since this paper was sent to press, I have made numerous observations regarding the therapeutic actions of hyoscyamine. In subduing the excitement associated with organic brain disease, in controlling simple mania, and even in the arrest of fits in the epileptic *status*, I have found it very efficacious. The result of these observations will be subsequently published. In the meantime it may be said that the drug produces great dryness of the skin. In a case of epilepsy in which, during the fits, the body is usually bathed with perspiration, the skin remains perfectly dry during the convulsions which occur previous to the full development of the action of hyoscyamine on the nerve centres.

excitement in which treatment by opium is contra-indicated, hyoscyamus and its active principle have been used with benefit. I have especially noticed that after the development of the full cerebral symptoms of the latter, when administered for the sake of determining the physiological action, a prolonged period of sedation follows, in which ideation is more than usually slow and a tendency to avoid muscular exertion is strongly manifested. Such a condition established for a short period in a case of acute or chronic mania, would place the disease under circumstances favourable to the restoration of normal mental activity. Still the therapeutic actions of hyoscyamus and hyoscyamine, though substantially established with regard to their effects on the function of the sympathetic and the secondary changes due to increased arterial pressure, are still wanting in precision in almost everything that concerns the modification of cerebral excitement, or even the production of sleep. That these actions, however, are inherent in the drug, and that to a greater extent than in belladonna, is beyond all question. Yet the method of employing them in such a way as to secure the full benefit of the corrective changes which they induce in the functional activity of the cerebrum, remains to be determined by prolonged observation of the modifications of brain function which follow upon their administration in health and in disease.
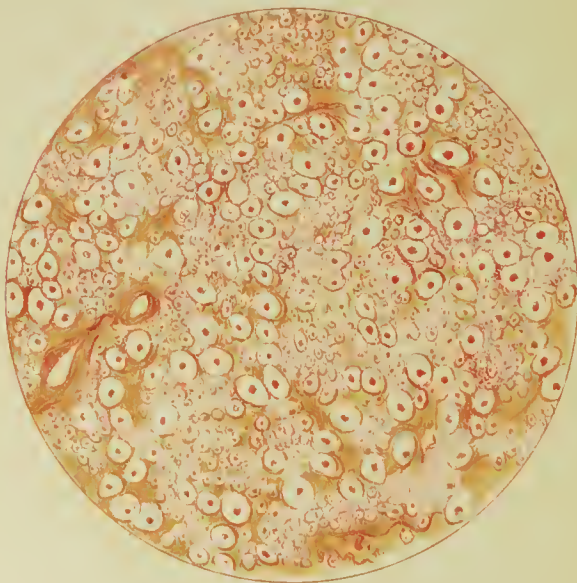
Fig 1.
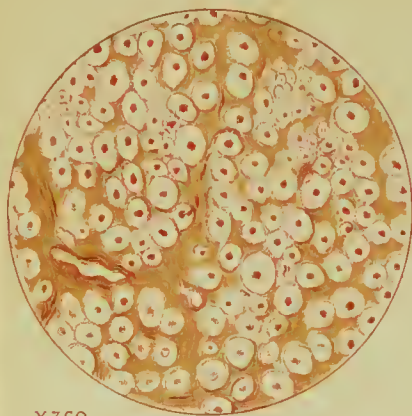
Fig 2.

X 350

X 90

Fig. 3.

X 350

Fig 4.

X 350

Fig 5.

X 350

ON THE HISTOLOGY OF THE GREAT SCIATIC NERVE

IN GENERAL PARALYSIS OF THE INSANE.

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AMONG the various paralytic lesions which interfere with the normal, equable, and regular transmission of nerve force to and from the sensorium, along the intricate network of the nervous system, none surely can claim our attention more justly than those dependent on the progress of that mysterious and fatal malady, the general paralysis of the insane. Prompted by the interest which such a subject arouses in the minds of all who take any interest in the obscure operations of disease within the *penetralia* of the nervous centres and their extensions, I have made it the special object of this article to bring under notice a few of the changes which may be observed in the nerves supplying the voluntary muscles of those who have died from this affection. In following up this investigation, attention has been more immediately directed to the healthy and morbid conditions of the great sciatic nerve, as its extensive distribution, combined with the fact of the serious loco-motor derangements existing in the limbs of general paralytics, warranted the assumption that any abnormal changes in the peripheral nerves, to which debility, spasm, or paralysis of the lower extremities might in some measure be due, would probably be detected in the trunk of this great nerve. Of all the forms of paralysing lesions which fall within the domain of the physician, the wasting, pro-

gressive forms are universally admitted to demand the most
serious prognostic judgment, and pre-eminently does this
folie paralytique deserve priority as the most intractable and
malignant in progress and termination of all varieties of
wasting palsy.   From the third decade of the present
century, when this disease first attracted the attention of
Bayle, Calmeil, and other alienists of the French school, it
has up to the present period formed the subject of constant
investigation by some of the greatest minds of the Eng-
lish, German, and French schools of medicine.   It is there-
fore with a certain amount of diffidence that I approach such
a subject, and advance my own views regarding the morbid
changes to which the cerebro-spinal nerves are subjected
by the ravages of this affection, although from numerous
and repeated observations I have been enabled fully to
confirm the opinion that there is some noteworthy morbid
change in the peripheral nerves of general paralytics, an
opinion first formed by Dr. Herbert Major, from casual
naked-eye examination of the sciatic nerve of a patient who
had died of general paralysis.   Before entering upon the
details of the morbid histology of the nerves, it will prove
advantageous to pass in rapid review the normal structure
and microscopic anatomy of one of the larger cerebro-spinal
nerves.   A transverse section of one of these large trunks
exhibits numerous funiculi, or bundles of nerve-fibres, bound
together by a common fibrous sheath or perineurium.   The
connective tissue forming this outer sheath is dense and
elastic, the latter property being dependent on the large
amount of yellow elastic fibres in its composition, and its
areolar extensions bind the funiculi in larger or smaller
bundles, and can be traced between the individual funiculi,
forming for them a delicate, elastic, but substantial frame-
work for support.   The whole of this fibrous investment
just described is by most authorities termed the neuri-
lemma.   Each funiculus has a special sheath in the form of
an extremely fine, homogeneous tube, sometimes slightly
reticulated, and separating the nerve-fibres from the
fibrous prolongations of neurilemma.   The nerve-fibres are
of course of the medullated variety, except in a few of the

cerebro-spinal nerves, where we occasionally meet with a small number of grey fibres also. They are usually round in outline, exhibiting in transverse sections the medullary sheath or white matter of Schwann, and the axis-cylinder or band of Remak. The latter structure is deeply dyed by carmine, logwood, or aniline-red, and is by this means made a very conspicuous object. It is of a solid, albuminoid composition, supported by the oleo-albuminous liquid around—the white matter of Schwann; which in its turn is limited by a delicate, homogeneous, tubular sheath very difficult of demonstration. This *primitive sheath* of the nerve-fibre exhibits, in a specimen carefully teased out and stained with carmine, nuclei attached along its whole course. The nerve-fibres of the great sciatic have been found to have an average diameter of ·012mm., but here and there scattered amongst the larger fibres within the funiculus minute medullated fibres, not exceeding ·002mm. in diameter have been seen. It appears that in the nerves of voluntary muscles the proportion of small to large fibres is very small, being about one to ten (Volkmann and Bidder). The individual funiculi in their course within the perineurial sheath blend at intervals with each other, and again divide, and do not form, what at first sight appears probable, single continuous tubes along their whole extent; but, on the other hand, they admit of extremely free communication *inter se*, so as to form a distinct plexiform arrangement, and in their coalescence allow a free interchange of the ultimate fibres from one funiculus to another. The nerve-fibre is assumed to retain its perfect continuity throughout, from its origin in the ganglion cell of the grey matter of the cerebro-spinal centre to its final distribution, where at length the axis-cylinder splits up into a fine network of pale, nucleated fibrillæ. The vascular supply of a nerve-trunk consists of numerous fine capillaries, which run parallel with the direction of its fibres, and entering the funiculi take the same direction there also. These longitudinal vessels are connected by transverse branches, diameter, of which, according to Henlé, hardly exceeds $\frac{1}{6000}$th of an inch. Having thus taken a brief survey of the histological

elements which enter into the formation of a nerve-trunk, their arrangement and appearance in a state of health as the conductors or internuncial agents of the cerebrospinal system, I proceed to note the morbid appearances met with in cases of general paralysis.

The sciatic nerve in general paralysis.—In all cases which I have up to the present time examined, this nerve has lost the firm, rounded contour which is possessed by it in the healthy state, has assumed an oval or almost flattened outline, and has been decidedly smaller, softer, and more pressible than in the normal condition.

Neurilemma.—The fibrous sheath is of a dull, dead, white aspect, and is almost entirely devoid of the beautiful, lustrous white appearance of the normal fibrous tissue; beyond this, however, no further morbid change is perceptible, either in the quality or quantity of connective element, as it consists of the usual amount of white and yellow fibrous tissue, which exhibits the usual nuclei upon the addition of acetic acid to the white element, the latter also appearing as wavy bands, streaked longitudinally as in healthy conditions.   The extension of this fibrous sheath inwards brings to view no abnormal changes.   The total amount of neurilemma appears not to be increased in quantity, the funiculi are bound together closely as in health, and the connecting fibres are well stained by carmine.

The funiculus is most readily detached from its bundles of nerve tubuli, forming a homogeneous tube, susceptible of being stained by carmine.   The average diameter of these funiculi has been found after numerous measurements to be somewhat reduced, but never to the extreme degree that is observed in many cases of senile atrophy.

The nerve tubuli.—Here the appearances become most significant of some pathological condition of great import. Extensive atrophy of the nerve tubuli is very apparent, but what immediately strikes the attention as being most peculiar and interesting, is the special course which this atrophy has pursued.   The wasting is not generally and equally diffused throughout the whole contents of the funiculus, but is limited to distinct regions, a bundle of nerve-

fibres appearing here and there to have taken on these morbid conditions, leaving the surrounding fibres perfectly healthy. Thus from *numerous* and *distinct* foci the pathological changes start *simultaneously* (as far as can be judged by the progress which the atrophy has made in each diseased bundle of nerve tubuli) and spread centrifugally, and do not, as in the more simple forms of wasting to be again alluded to, cause an equable diminution in size of all the contained elements of the funiculus. It must be remembered, however, that the affection which we are now considering is especially gradual and progressive in character, and hence we find, what we should *primá facie* expect, that the varied stages of atrophy, in its onset, progress, and complete ascendency, predominate in the several funiculi of the nerve-trunk. Thus when a transverse section is examined by a low power, we find numerous funiculi exhibiting a peculiar punctated appearance from a deeper and more general dyeing of these points by carmine; whilst others show large patches of the same character, involving the more healthy portions around to a far greater extent; and in still further advanced atrophy, two or more of these centres of pathological change have united so as to form one irregular bundle of wasted tubuli. It is by the progressive character which these morbid changes therefore assume, that the opportunity is afforded of recognising the earlier and later stages of atrophy of the nerve tubuli, when our patient has eked out his miserable existence to the extreme verge of the third stage, and has died in a state of utter fatuity. In no case have I seen the nerve-tubule so atrophied as to exhibit the axis-cylinder alone, for in the most minute fibres left the medullary sheath always remained, even though at times reduced to a mere white rim around the band of Remak. As the medullary sheath had become more and more wasted, so *pro rata* had the axis-cylinder dwindled away to a mere streak, yet still susceptible to staining by carmine. The staining with carmine was peculiar, for in all cases I failed to obtain the deep equable coloration of the nerve axis which is so readily obtainable with sections of the healthy nerve.

The vascular supply.—The capillary networks, which in the normal state form by the transverse branches before alluded to, numerous oval or elongated meshes become apparently more numerous and enlarged. There is every evidence left of an increased vascularity of the nerve during life, the nerve-tubes are closely surrounded by vessels, often widely separated the one from the other by the intervention of tortuous, distended channels, whilst the vessels running parallel to the direction of the nerve-tubes, as seen in transverse section, are unusually large and coarse. It is believed by some histologists that some of the elements of connective tissue are to be found amongst the nerve tubuli within the funiculi. One of our best authorities says on this point:—
'It has often appeared to me as if there were filaments of extreme tenuity, like the white filaments of connective tissue, but of doubtful nature, mixed up with well characterised nerve-fibres within the sheaths of the funiculi.'[1] But, although it is difficult to demonstrate the existence of these microscopic threads as a normal constituent of the funiculi, yet the presence of connective tissue in this situation can be readily shown as one of the results of those pathological changes connected with the complex morbid phenomena of general paralysis. I have been able clearly to demonstrate the presence of a tolerably thick web of connective tissue between the capillary network and the nerve-tubules; the connective cells are mostly stellate and their prolongations very fine. They may be readily shown in a funiculus deprived of its sheath, recently obtained, and teased out carefully and stained with carmine. Whence does this overgrowth of connective tissue proceed? In considering this question, which has very important bearings, it must be remembered that the endothelial cells of capillary vessels are strictly representatives of the stellate connective tissue corpuscles, and that the latter may even replace the former in what Rindfleisch terms his 'tertiary mode of vascularisation.' This last-named author moreover affirms that 'the evidence as to the homology of the connective tissue corpuscles with the cells of the endothelium is completed by the

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[1] Quain's 'Elements of Anatomy,' 7th edition, p. cxlv.

homology of their formative products, products which both are capable of yielding in a certain measure.' Increased vascularisation, together with a well-developed, easily demonstrable web of connective tissue as a morbid accompaniment of general paralysis within the sheath of the funiculus, affords strong evidence in support of Professor Sharpey's views in regard to the existence and nature of the minute fibres described by him. A full confirmation of these views appears to me especially important in the wasting diseases of the peripheral nerves, as they carry with them a pathological significance of the highest value in the elucidation of all such morbid processes. To sum up then in brief terms the morbid lesions detected in the nerve-trunks of general paralytics, we have :—

1. Funiculi greatly diminished in size.

2. A peculiar fasciculate atrophy of nerve tubuli, involving both medullary sheath and axis-cylinder.

3. Non-susceptibility of the axis-cylinder to normal, deep staining by carmine.

4. Increased vascularisation.

5. Hyperplasia of the intra-funicular connective element.

Having thus taken a brief survey of the morbid lesions occurring in the nerves of general paralytics, it may prove instructive to turn our attention to the more frequent and simpler forms of atrophy occasioned by senile decay, or by disuse of a limb from accident or deformity.

Senile atrophy.—The sciatic is always found greatly wasted, extremely soft, very readily torn up, and communicating a greasy, unctuous feeling to the fingers from the predominance of adipose tissue.

Neurilemma.—This component of the nerve is much altered in appearance, both in its formation of the fibrous, perineurial sheath and the bands of connective tissue which normally separate the funiculi from one another. The yellow elastic element is almost entirely absent in some cases, whilst in others its amount is decidedly far below the average of the healthy standard. The wavy bands of white fibrous tissue no longer exhibit their characteristic forms, but are represented by numerous short fusiform bands very

loosely connected together, and scattered amidst an abnormally large supply of adipose tissue. The peculiar character of this degenerate form of connective tissue renders it a most ineffectual support for the nerve funiculi.

The funiculi.—All are usually greatly reduced in size, and very loosely bound together, and in most cases the advanced stage of atrophy, together with the depraved form of the connective medium, has allowed the funiculi to recede far from the neurilemma, leaving wide interspaces often filled with adipose tissue. The larger funiculi are frequently found split up into numerous small, irregular compartments by the enlarged blood-vessels and the hyperplasia of connective tissue.

The nerve tubuli.—It is a rare matter to find a few average-sized tubuli, so extensive is the atrophy in cases of senile dementia. Nor, on the other hand, has the morbid process attacked certain series of nerve tubuli successively, for there is no appearance in the slightest degree resembling what has just been described as so characteristic in cases of general paralysis. There is uniform and general atrophy of all the tubuli collectively, with much alteration in the contour of the tubule, probably dependent on an abnormal condition of the white matter of Schwann, together with changes proceeding in the primary sheath. The axis-cylinder always takes the carmine or aniline staining rather feebly.

Vascular and connective elements.—In transverse sections the calibre of the blood-vessels is found greatly encroached upon by increase and hypertrophy of their coats, and this condition has reference both to the vessels outside and inside the sheath of the funiculi. The capillary network within the sheath, and in the conditions of health, is one of great delicacy, the individual capillaries often not exceeding $\frac{1}{6000}$th of an inch in diameter, but in the morbid state now under consideration their diameter is greatly varied, being narrowed and often obliterated at some points, swollen and tortuous at others. The larger vessels always exhibit a narrowing of their lumen, as seen in transverse section, together with an unusually large supply of the oblong contractile cells of plain muscular tissue and hypertrophy of the tunica adventitia.

Fatty degeneration has also left its stamp upon the contents of the funiculus, oil globules and granular *débris* being found here abundantly, as also in the neurilemma around. Great increase in the tunica adventitia of these capillaries appears to have been the starting-point for the general increase of the connective tissue within the funiculus, and which encroaches greatly upon the nerve tubuli.

The morbid appearances above described in the nerves of those who have been the subjects of senile atrophy are also met with in all cases, where from long-standing disuse of a limb, and many other causes interfering with the due functional activity of the part, the usual amount of nervous energy conducted along its various strands has been limited greatly, or entirely arrested. The conditions therefore dependent upon this diminution of nervous activity may be summed up as follows :—

1. Degeneration of connective tissue.

2. Funiculi greatly lessened in size, and receding from their neurilemmal investments.

3. General diffused atrophy of nerve tubuli.

4. Proliferation of connective tissue within the funiculus with sclerosis of the arterioles and capillaries.

5. General fatty disintegration.

I have intentionally relegated to the concluding portion of my paper any views which I hold concerning the origin and production of the peculiar motory phenomena of general paralysis, as these views are based exclusively upon the morbid post mortem appearances of the nerves and their reaction, in its various stages, to the stimulus of the induced current. Upon careful comparison of the foregoing details, for the purpose of a more thorough appreciation of their pathological import and bearing, it appears evident that the changes recorded in those who were the subjects of senile atrophy formed but one link of that chain of morbid processes so well recognised as occurring in the tissues on the advance of senile decay. It appears that coincident with the degeneration of the cells and capillaries of the cerebral cortex, the atheromatous conditions of the vascular system and degeneration of the muscular contractile fibre, come the selfsame

changes in the peripheral nerves—the formation of a degene-
rate type of tissues, with their ultimate breaking down into
fatty and granular *débris.* These simpler forms of nerve
atrophy appear dependent for their causation on the exces-
sive growth of connective tissue around the arterioles and
nerve-fibres, which exerts direct pressure upon the latter just
as it limits the flow of nutrient fluid in the former. Two
other elements of morbid action must, however, be superadded
to the constrictive effects of this hypertrophy, viz., the en-
feebled and greatly diminished supply of nervous energy,
together with an unnatural condition of the nutritive fluids.

Now it appears to me that the indications met with in
the nerves of general paralytics as regards the connective ele-
ment, point chiefly to a non-inflammatory hyperplasia of
this tissue as the result of persistent dilatation of the vessels
—a passive congestion due probably to vaso-motor paralysis.
The protoplasmic masses derived from these engorged vessels
infiltrate the tissues, become transformed into connective
tissue, whilst the abnormal amount of nutrient material gives
rise to overgrowth and hypertrophy of the already existing
representatives of this tissue described by Sharpey. Yet it
must not be supposed that this proliferation of connective
tissue proceeds to any very great extent in the peripheral nerves
as it does in the atrophy dependent on senile changes; in
fact, the nerve tubuli are comparatively, but very slightly, en-
croached upon, and the wasting of distinct strands of tubuli,
often most evident at places where there is least develop-
ment of this tissue, tends to confirm my conclusions that this
hyperplasia is not the cause but simply a concurrent change
of the atrophy in the nerve tubuli. I term the changes ob-
served in connective tissue of the funiculi a 'non-inflamma-
tory hyperplasia,' and I believe the appearances thoroughly
warrant this assumption, for in no single case of this disease
could I detect the evidences of a previous inflammatory
action. The remarkable changes which occur in nerve-tubes
in inflammation of their structure have been lately made the
subject of special investigation by Mr. Hamilton, at Vienna,
in the examination of the cord in artificially induced
myelitis. It appears from his researches that oval swellings

occur along the course of the axis-cylinder, often five to ten times its normal diameter, of a transparent substance yet contained within an attenuated, distended nerve-sheath, and deeply stained by carmine. These oval swellings appear subject to fissiparous division, and invade the surrounding neighbourhood by their escape from the nerve-sheath, becoming identical with the well-known colloid bodies, and, in the seat of most intense inflammatory changes, to granular degeneration, with the formation of pus-cells in their interior. Swelling and molecular transformation of the nerve-cells, with very slight alteration in the neuroglia, and the presence of numerous leucocytes in the vascular sheaths, ccmplete the changes produced in myelitis.[1] Such therefore being the morbid effects on the nerve-tubes in inflammatory affections of the cord, we are warranted in assuming the changes in general paralysis to be those of passive congestion and purely non-inflammatory hyperplasia. The distinction which I have thus thought proper to draw between these non-inflammatory overgrowths and the actual state of inflammation of nerve-tissues, is more for scientific accuracy than for any practical deductions, as I readily admit that under certain conditions both groups merge into one another, and no actual line of demarcation broadly separates them. In turning our attention to the wasted appearances of the nerve tubuli, it is essential that a proper understanding should exist as to the conditions of electro-muscular excitability, of reflex action and cutaneous sensibility, and it will, I think, be found that such an investigation throws much light upon the nature of the special form of atrophy we are at present considering. The electro-excitability of the muscular system in general paralysis, and other nervous affections, has been made the special subject of investigation by Mr. Lowe, who published the results obtained by him in the third volume of the West Riding Asylum Reports, and to which I would direct the attention of the reader. Drs. Bucknill and Tuke have also stated their views in regard to this matter in the last edition of their ' Psychological Medicine.' I have made a series of independent observations on

[1] The ' Monthly Microscopical Journal,' No. 79, p. 41.

this point, and the results so entirely accord with the views of
these writers, that I need not give them here *in extenso ;* yet,
as Mr. Lowe has not made special mention of more than two
or three muscles of the lower limb in general paralysis, I
think it may be desirable to state that the results of my ob-
servations tend to prove that electro-muscular excitability is
retained for a much longer period in the flexors of the leg
and extensors of the foot, than in the flexors of the foot.   No
response whatever could be obtained from the peroneii, the
extensor longus pollicis, extensor communis digitorum, and
tibialis anticus, except by an extremely strong current, and
even in this case the extensor pollicis and tibialis anticus did
not contract.   The flexors of the toes responded, though
rather feebly ; but the electrodes immediately produced
strong and active contraction in the soleus, gastrocnemius,
and tibialis posticus.   .

Reflex action of the lower extremities was extremely
deficient, and in several cases almost totally abolished.
Cutaneous sensibility also was very greatly lessened in the
later stages of general paralysis, but was most acute along
the inner side of the foot.   The following table exhibits a
few results obtained from the cases examined to illustrate
these points.   In each individual excito-motory action, mus-
cular excitability, together with cutaneous sensibility, was
thoroughly examined by pricking with a needle, tickling of
the soles, or the application of the electrodes of Störher's
larger battery.

Case	Reflex Action	Cutaneous Sensibility
E. R.	No response.	Almost absent.
M. M.	Extremely feeble response.	Very deficient.
E. S.	do.	do.
M. G.	do.	do.
M. W.	No response.	do.
M. B.	Feeble response.	Normal.
F. H.	do.	Very deficient.
T. B.	do.	do.
M. A.[1]	Normal reaction.	Normal.
M. B.	do.	Acute.
H. G.	do.	do.

[1] These last three cases were admitted with symptoms closely allied to those
of general paralysis ; two of them have since turned out, however, to be not so,
and one still appears doubtful and anomalous.

Thus it may be stated, as a well-ascertained fact, that in the later stages of general paralysis the general cutaneous sensibility of the lower extremity is greatly reduced, the general reflex actions are diminished or abolished, and special sets of muscles respond feebly or not at all to the stimulus of faradisation. This diminished excito-motory sensibility has been noticed in the last edition of their work on Psychological Medicine by Drs. Bucknill and Tuke. A series of experiments illustrating the valuable diagnostic indications afforded by this means were instituted by Dr. Bucknill in the year 1851. The first of these conditions points to some lesion in the afferent nerves, or in some portion of the sensory tract. Whether the site of this obstruction to the flow of sensory impressions upwards be the intra-cranial ganglionic system, the spinal axis, or the afferent nerve-fibres of the cord, cannot yet, I fear, be determined with absolute certainty. It appears, however, highly probable that this cutaneous anæsthesia depends upon disease of the posterior columns of the cord propagated down the sensory nerves ; and I have little doubt that many of the atrophied tubuli are really the remains of wasted *afferent* fibres. In the earlier stages of this disease, the whole sensory tract is in a state of exalted functional activity, and cutaneous hyperæsthesia is the result of this condition. The slightest prick with a pin over the integument of the foot, the slightest pinch, will produce an exclamation of pain, and tickling of the soles of the feet appears more than usually unbearable. This state, however, very soon subsides, and the loss of sensitiveness becomes very marked as morbid changes affecting the sensory tract proceed ; and yet it would appear more probable that this sudden change was due to lesions in the conducting strands of fibres, rather than in the more central portions of the nervous system and their ganglionic cells. A sudden movement will often make the general paralytic spring back with an aspect of terror; a sudden sound will startle him and cause much agitation ; even addressing him unexpectedly appears in these last stages of the disease frequently to propagate a shock to the hyper-sensitive auditory ganglia, which may leave the patient

trembling like an aspen-leaf. The sensory ganglia conse-
quently are maintained in this abnormally acute and
sensitive condition, even through the last scenes of this
fearful malady; and we are therefore driven to seek for an
explanation of the cutaneous anæsthesia in the sensory
columns of the cord, or the peripheral nerves themselves.
The changes which are usually found in the cord are over-
growth of connective tissue in the posterior and postero-
lateral columns, giving rise to extensive atrophy of nerve-
tubes in these situations, the morbid process often extending
from the cervical into the lumbar regions. This pathological
condition would of itself account for the abnormal relation-
ship existing between the lower extremity and the reflex
functions of the spinal cord; yet it appears to me highly
probable that the afferent fibres of the spinal nerves are
atrophied as a result of the same condition. If we now turn
our attention to the locomotor derangements of general
paralysis—the peculiar gait, the ataxic movements of the
limbs, &c., I believe there exists evidence which suffices to
establish one important fact—viz., that these irregular
spasmodic motor derangements are due, not so much to the
implications of the afferent sensory tracts, as to direct
lesions of the efferent nerve-fibres. It is a well-known phy-
siological truth that in certain conditions, where the will is
powerless and utterly incapable of calling forth any special
voluntary muscular action, the same action has been readily
produced through the medium of the emotional centres. In
the case of F. H. (*vide* table) locomotion was much im-
paired, the gait being more of the paralytic than the ataxic
character, when suddenly the patient became greatly excited,
torpor and apathy vanished, dementia gave place to decided
temporary mental improvement, and all the exalted delu-
sions of a general paralytic in the most characteristic
stage, burst from her tremulous lips, the countenance
being expressive of the ' ruling passion strong in death.'
But this sudden fit of excitement brought with it peculiar
modifications of the locomotor derangements. No longer
could the gait be characterised as the paralytic variety de-
scribed by Westphal, but it assumed the tabic type as regards

the high lifting of the foot and outward jerky movement; and, excepting the fact that the patient was capable of standing firmly, even with the eyes closed, the condition very closely resembled that of locomotor ataxy. The patient was able to walk about with very little trouble, and was apparently delighted with her newly-acquired powers. This case exhibited most decided loss of reflex spinal action; the cutaneous sensibility was at an extremely low ebb, and when the sole of the foot was tickled no withdrawal of the part occurred; but, on calling her attention to the act and directing her eyes to her feet, the slightest attempt at tickling made her exceedingly emotional, and occasioned convulsive efforts to retract the leg. This is but a single illustration of what may be constantly seen in cases of general paralysis and other neuroses pointing to analogous pathological lesions, and the explanation of such phenomena is clearly this: that the channel whereby the reflex spinal currents were wont to travel having become degenerated, these irregular ataxic movements, produced by the higher functional manifestations of the central ganglia and cortex, indicate a progressive change in the motor tract or the efferent fibres of the spinal nerves. The peculiar gait noticed in most of these cases, the spasmodic movements induced by volitional impulses, are clearly due to a true ataxia, a failure in the harmonious actions of co-ordinate groups of muscles. One other form of progressive paralysis shares in this peculiarity of irregular muscular movement. I refer to locomotor ataxy, and in both affections the lesions met with in the cord are similar, and consist essentially in a posterior spinal sclerosis. The explanation which instantly occurred to me of these derangements of movement, after a cautious examination of the morbid histology of the nerves in general paralysis, is one which I afterwards discovered had already been employed by Dr. Patrick Nicol, in an excellent article on the 'Pathology of Locomotor Ataxia,' in the West Riding Asylum Reports for 1871. Dr. Nicol there assumes for locomotor ataxia a condition in every respect identical with that which appeared to me from the first as the most plausible and rational explanation of the ataxic phenomena of general paralysis.

In the latter affection we clearly have an atrophy or blocking-up of certain channels whereby the transmission of motor impulses is interfered with. What is the most natural result of this partial interference? The amount of nervous energy developed in the ganglion cell becomes impeded in its flow down the atrophied tubuli, and its intensity remaining as in the normal state of functional activity, an overflow must take place through those channels which retain their normal conductibility. For it must be remembered that the demand upon the cineritious substance for each individual movement is governed entirely by prior experiences and early education of the ganglion cell. Thus would I seek to explain those exaggerated contractions of individual muscles, those irregular spasmodic twitchings, occasioning the jerking movements of the limbs, displaying the striking effects of the introduction of such discordant elements amongst the co-ordinate groups of muscles. The harmonious action of each group is prevented by this occlusion of certain channels of innervation, and the increased collateral circulation of nervous energy by this means established. The varied nature of the nerve-fibres in the afferent or sensory roots still remains a *quæstio vexata* to physiologists. Nerves of muscular tone, nerves of tactile and common sensibility, of reflex action, and thermic nerves, have been spoken of; but whatever be their varied character, it appears certain that the due activity of muscular sense is essential to the maintenance of muscular tone, and that when the former is by total arrest of circulation abolished, total loss of muscular power ensues; so also intense cold, by diminishing the sensibility of the integument, will greatly modify muscular activity. Hence we have every reason to infer that atrophic changes in the cells or tubuli of the sensory tract must be followed by a diminished tonicity of the muscles which are brought into relation with the diseased parts. In extreme cases, when muscular sense is abolished, as in locomotor ataxy, the muscular power is no longer subject to the directive influence of the will, but sight is essential to the accomplishment of any individual volitional movement of the limbs, supplying the guiding principle normally presented by the muscular sense. In general

paralysis the implication of the sensory cells does not appear to be so extensive as in locomotor ataxy; yet to this particular lesion may partially be attributable the ataxia. Loss of tone in a special series of muscles may be one element in the causation of irregular muscular actions, yet the more important factor appears to me to be the establishment of a collateral circulation of nervous force.

It seems to me highly probable that in locomotor ataxy the afferent sensory tract is far more gravely implicated than it is in general paralysis, and that though in the latter affection both sensory and motor strands suffer, yet the lesions are far more decided and serious in the efferent fibres and their ganglion cells. The more closely the lesions of general paralysis are subjected to critical examination, the more certain does it become that the pathogenesis of this obscure malady must be sought for in some general vice of the nutrition of the nervous system, and most significant therefore does the extensive vaso-motor paralysis become in its bearing upon this question. The conditions upon which this general paresis of the vaso-motor system depends merits close consideration, and may yet elucidate more clearly than any other condition the true pathology of many obscure affections.

It is a well-known fact that if the sciatic nerve of an animal be severed the resulting phenomena are very marked; thus extensive vaso-motor paralysis ensues, the blood-vessels of the corresponding limb widely dilate, and a rapid elevation of temperature occurs in the paralysed limb, which often ranges 10° to 15° above that of the sound member. The same result is observed upon section of the spinal cord through the lower part of the lumbar enlargement. This experiment is therefore equivalent to Bernard's section of the cervical sympathetic, being due to the division of the vaso-motor centres and their efferent branches, for the spinal nerves convey these filaments in their interior, deriving them not only directly from the cord, but in their course subsequent to their emergence from the spinal centre. Now it has been ascertained by Professor Goltz, that on dividing the lumbar enlargement of the cord, and permitting the ensuing vaso-motor paresis to subside, in a few days we may,

by stimulation of the severed extremity, reproduce the same condition—viz., distinct dilatation of the vessels supplying the limbs, together with decided rise of temperature.   So also on applying the faradic current to the proximal extremity of a divided sciatic nerve the temperature of the opposite limb rose several degrees.   In these cases the stimuli applied were mechanical, electric, and chemical.   The same authority also states that on irritating the peripheral end of the divided nerve a rise of temperature ensued, but it appears to me that these last observations admit of great doubt, not only on account of the very trivial elevation of temperature ensuing, but faradisation being chiefly employed as the irritating medium, the muscles were tetanised, and hence fallacies naturally arose. It has been urged on the consideration of physiologists by Professor Goltz that these results of his establish a very strong argument in favour of the existence of active dilating vaso-motor centres and filaments.   But it will at once be apparent to the reader that, if the vascular dilatation and thermal changes were due to irritation of an active dilating vaso-motor centre and of its branches, as presumed to exist by Professor Goltz, then no probable explanation could be given of the fact so well established by Bernard—that diminution of the vascular calibre occurred immediately on irritating the divided extremity of the cervical sympathetic. Professor Goltz attempts to explain away this important discrepancy by reference to the complicated connections of the cervical ganglia, and the comparatively simple arrangement obtained in the sciatic trunk.   I do not think this view of the case, however, can possibly invalidate the results of Bernard's experiments, as irritation was in them applied more directly to the sympathetic trunk than in the experiments of Professor Goltz.   It certainly appears to me far from a necessary deduction that we should presume upon the existence of these hypothetical dilating nerves of the vaso-motor system.   What then appears the rational explanation of these experiments of Professor Goltz?   Clearly, I think, the explanation involves the supposition that numerous vaso-inhibitory centres exist in the cord, and that these are in *close connection with the spinal centres of the afferent or sensory*

nerves. Innumerable facts might be brought forward in favour of the doctrine that the spinal cord includes numerous centres of vaso-inhibitory action, which upon the receipt of impulses from the nerves of common sensation react upon the vaso-motor filaments, and produce by inhibition of their ordinary functional manifestations a corresponding dilatation of the arterial walls. Take a case in point—a reflex muscular movement of a limb from some sudden irritating cause, say a scald. Here we get the impression travelling along the ordinary route of excito-motor actions exciting an impulse in the motor centres of the cord, which conveyed along the efferent nerve-fibres produces the contraction of a group of muscles, and the removal of the limb from further injury. But this is not all: the irritation received by the peripheral distribution of sensory nerves conveyed upwards *inhibits* within the spine various vaso-motor centres, causes a dilatation of the vessels at the seat of injury and those supplying the active muscles. In the contraction of every muscular effort it appears probable that the energy evolved by the nervous centres for the accomplishment of this object must necessarily increase by inhibition of the vaso-motor centres the vascular supply of the muscle, and that this inhibitory action is graduated by the energy of the muscular act varying in direct ratio with the force of the contraction may, I think, be assumed on very rational grounds. To revert to our more immediate subject, let us try to apply these principles to the lesions of general paralysis. The atrophy of the nerve is by no means a necessary sequence of the vaso-motor paralysis, for cases are on record of extensive congestions dependent on injury or disease of the vaso-motor system, without any well-marked nutritive changes in the tissues; nor is there any evidence which clearly proves that mere alteration in calibre of the blood-vessels of a part, through the influence of this system of nerves, can modify to any great extent the general nutrition of the tissues. It appears more rational to regard the wasting of the nerve-fibres in this disease as probably the partial cause of vaso-motor paresis, rather than a secondary result of the changes in this system, as the progressive changes in the sensory

roots and the cells of motor and sensory centres in the cord would necessarily involve these assumed centres of vaso-motor inhibition. The trophic disorders in the muscles of general paralytics bear no relationship to the vaso-motor changes, but appear to me as probably dependent upon the atrophy of the cells and nerves of reflex-spinal action. That there are special centres in the cord for the maintenance of a trophic nervous influence on the body is an opinion which has many adherents. The peculiar pathological changes observed in the spinal cords of those who have suffered from progressive muscular atrophy, in which disappearance of numerous cells from the anterior cornua is coincident with paralysis of a *special class* of muscles without diminution or modification of cutaneous sensibility, appear to me as very decided evidence in support of the theory of trophic nervous action, and the close connection between these trophic centres and the motorial centres of the cord. Hence it is that I feel justified in concluding that in general paralysis of the insane, the extensive implication of afferent and efferent fibres of excito-motor tracts, together with changes in their sensory and motor spinal cells, cannot but affect through their intimate relationships the trophic and vaso-motor centres of this region.

ON TEMPORARY MENTAL DISORDERS AFTER EPILEPTIC PAROXYSMS.

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In the wards of a general hospital we meet with many cases of insanity—mostly temporary. For example, patients are brought in by their friends or by the police for sudden attacks of mental disorder occurring after epileptic fits. Then there are cases of severe wounds, self-inflicted by lunatics; and, of course, temporary mental disorders occasionally occur in patients who have been some time in the hospital for non-mental diseases. Besides, there are cases of delirium tremens, and all degrees of delirium in patients with acute disease—rheumatic fever, pneumonia, erysipelas, &c. So far I have spoken of active or positive mental symptoms. In cases of intra-cranial tumour, cerebral hæmorrhage, softening, &c., all grades of the negative condition of imbecility are seen.

At this moment [1] there is in the hospital a patient who is maniacal after severe injury to the head and erysipelas. There · is a woman maniacal of whose antecedent history nothing certain is known, but from her high temperature, from the state of her tongue, and the nature of her stools, she is supposed to be suffering from typhoid fever. [So it turned out.] The woman came herself to the hospital saying that she had had a 'fit;' possibly it was simple fainting. There is another maniacal woman who may possibly be just

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[1] When this was written.

recovering from typhoid fever, but of her history nothing is known. These patients are not simply delirious, but delirious with violent action—requiring restraint. A man is in the hospital who is just recovering from a self-inflicted wound in the throat; he is subject to slight fits, and his account that he knows nothing of the infliction of the injury is credible.

I think then the study of mental diseases may be well *begun* in general hospitals. We have there the advantage of seeing transient and very slight mental disorders—simple cases which are comparatively easily studied. Especially do we see or hear of many degrees of slight and transient disorders of mind after epileptic paroxysms, in the wards and in the out-patient room.

There are few diseases of more practical interest than epilepsy. Reynolds says that seven per cent. of nervous diseases are epileptic. Besides this and other good reasons for the assertion, there is the fact that epilepsy is often associated with insanity. Indeed, according to Bucknill and Tuke, six per cent. of persons in asylums owe their insanity to epilepsy. It is not asserted merely that six per cent. of insane patients have epileptic fits, but that epilepsy is the cause of insanity in six per cent. of insane persons.

We have even stronger grounds for the assertion. The insanity of epileptics is often of a kind which brings them in conflict with the law. We have not only to treat epileptic patients, but we have occasionally to declare whether an epileptic is or is not responsible for certain quasi-criminal actions. The epileptic is beset with troubles; besides the calamity of fits, and besides such ill chances as severely burning his face by falling into the fire, there is the possible further calamity that he may be punished for 'crimes' he has committed unconsciously directly after a seizure. We may help them here as well as by treatment of their malady.

Epileptic insanity is usually violent, and the violence may take the form of crime from purely accidental circumstances. Falret says that all authors have noted the excessive violence of the acts of epileptic maniacs. I suppose this remark is made of asylum cases, of patients so bad that they require continued restraint.

Not being an alienist physician, I mostly see cases in

which the mental symptoms after epileptic attacks are comparatively slight; often they are merely grotesque actions. I purposely dwell most on the slightest cases of all. I shall, I hope, give good reasons for doing so. In all the cases I relate, even in those where the symptoms were severe, the patients, prior to the outbreak of their temporary post-epileptic insanity, were in supposed good mental health; of their real mental condition I can know nothing accurately; at any rate, they were well enough to be at their occupations. A goodly number are brought to the hospital from workshops or from the street. Again, the symptoms in the cases I see are often very transitory.

Let me at once relate a case in order to show the kind of case of epileptic insanity we often see at the London Hospital, and the difficult circumstances under which we sometimes see it. I do not mind confessing that I have more than once found it hard to tell whether a patient's violent conduct was owing to an epileptic attack, to drunkenness, or to meningeal hæmorrhage. In some cases of fatal meningeal hæmorrhage, the patient is not apoplectic but uproarious. It is so also in some cases of severe and fatal injury to the head. We occasionally see maniacal patients without any history whatever; we sometimes cannot tell whether the patient has had a fit of any kind or not.

A man was brought to the hospital about six o'clock P.M., said to have had a fit, and to have been extremely violent. When first seen he was struggling with the police who had brought him in, but as soon as freed he appeared sensible but irritable. He dressed himself and looked about sensibly. He then attempted to leave the room, and on being prevented became very violent, hit out blindly, and had to be held down. After this he could not, or did not, give his name, and when asked his address said Bethnal Green. He asked for his bag (he was a postman), and was much disquieted until he got it, when he appeared thoroughly satisfied. Subsequently he repeated his attempt to leave, and his blind violence when prevented. An hour and a half later he gave his name and address, said that he was accustomed to have fits, and that on several occasions he had lost his senses for half an hour, or for several hours; at this time he said he felt

perfectly well again and fit for duty. His wife said that he was generally violent after the fits, that he had threatened her life, and that she was much afraid of him in the fits.

As I speak of epileptic insanity, I ought perhaps, before I go further, to say what I mean by Epilepsy ; for my opinions as to the nature of epilepsy are accepted by very few physicians. But for my present purpose I will adopt the accepted definition of epilepsy. I can properly do this, as the definition is symptomatic. The genuine epilepsy of authorities is but one epilepsy (according to my definition of the word epilepsy).

According to authorities, epilepsy is a chronic disease of which the characteristic symptom is a sudden loss or trouble of consciousness occurring occasionally. The affection of conciousness is sometimes accompanied by evident spasm of muscles, and sometimes occurs without obvious spasm. The essential thing, according to this definition, is the paroxysmal affection of consciousness. It should, however, I think, be the occurrence of affection of consciousness very early in the paroxysm.[1]

There are three varieties of epilepsy as it is above defined. This division of course is arbitrary, although it is convenient. In fact, these are not so much varieties as degrees of the same thing. The three are called (*a*) vertigo, (*b*) le petit-mal, (*c*) le grand-mal. Not only is there affection of consciousness in each of these, but it is, I repeat, the first, or nearly the first, thing in the paroxysm ; this must be borne

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[1] The distinction into cases of epilepsy with, and cases of epilepsy or epileptiform seizures without, loss or trouble of consciousness is useful for practical purposes. But, scientifically, it is arbitrary and has no rational basis. It is an empirical distinction of psychological, not of anatomical or physiological, parentage. The epilepsy of authorities is to me but *one* epilepsy ; the paroxysms in it are, I consider, results of 'discharging lesions' beginning in the *very highest* nervous centres of the cerebral hemisphere. Other epilepsies (such seizures, for example, as are commonly called epileptiform) differ in that in them the 'discharging lesion' is of nervous arrangements of subordinate centres in the cerebral hemisphere. In them consciousness is lost late or not at all. But even empirically and for practical purposes (that is, from the symptomatic or from the clinical point of view), the distinction should not be into genuine epilepsy and epileptiform seizures ; it should be into cases in which loss of consciousness is the first event and cases in which it occurs early, cases in which it occurs late, or not at all.

in mind. There are three other things about epilepsy to be kept vividly in mind.

(1) That there occur all degrees of obscuration of consciousness, not loss only, in cases of epilepsy. From temporary confusion of thought (which is 'defect of consciousness') to deepest coma there are all gradations. In fact, the patient who is subject to 'genuine' epileptic attacks may have had (what he will probably call 'sensations' or 'turns') abortive seizures without any affection of consciousness before he ever had a full attack; for example, he may have had paroxysmal epigastric sensations only.

(2) That there are all degrees of severity of epileptic paroxysms, from giddiness attended by trivial confusion of thought, to a full violent seizure with universal convulsion and deep coma. This is indeed almost a repetition of the former statement, and it is repeating that the three so-called varieties or degrees are really arbitrary separations.

(3) And, most important, that cases of epilepsy in which there are the slightest attacks are the worst for mind. We must not forget this for two reasons: (*a*) the importance of these attacks is unfortunately often underrated because they are slight; (*b*) the attack being slight and transitory it is liable to be overlooked, and attention may be paid exclusively to its sequelæ, that is, to the patient's grotesque actions, raving, &c. These may in a woman be erroneously attributed to hysteria, and in a savage man to criminal intent. The gravity of these cases is not because the paroxysms are slight, but because the 'discharging lesion,' in cases in which such slight fits often occur, is of the highest and most intellectual nervous arrangements (substrata of consciousness).

I intend to speak only of temporary insanity *after* epileptic paroxysms. There are two other ways in which mental disorder is related to epilepsy. There is not only (1) the sudden and transient mental disorder after one or a few fits, but also (2) more lengthy infirmity after a rapid succession of numerous fits; and again (3) the persistent deterioration (imbecility), the [1] result of fits repeated for months or years.

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[1] Falret concludes that 'delirium chiefly occurs as a consequence of epileptic attacks, recurring at short intervals after a prolonged suspension of the disease.'

Cases of epileptic insanity have, of course, been long well known to alienists. For the first, and also I believe the best, scientific account we are indebted to Falret. Trousseau has given an account of the subject in one of his lectures. The third part of Trousseau's lecture is made up almost entirely of quotations from Falret's book. A most valuable case of 'masked epilepsy' has been reported by Dr. Thorne Thorne, St. Bartholomew's Hospital Reports, 1870.

I intend to consider not only cases of violent doings, but cases in which the patient simply acts oddly, as, for example, such cases as that of a patient who after a paroxysm blew his nose on a piece of paper, cases in which there is no direct medico-legal interest. The latter have nevertheless an important *indirect* medico-legal interest. It is convenient to have one name for all kinds of doings after epileptic fits, from slight vagaries up to homicidal actions. They have one common character—*they are automatic;* they are done unconsciously, and the agent is irresponsible. Hence I use the term *mental automatism.* I say mental, as the doings are probably external signs of crude mental states,—external signs of 'epileptic dreams.'

Every one of the cases I have to relate is an illustration of Laycock's doctrine of Reflex Cerebral Action; in fact, I hope the cases will show that this hypothesis of Laycock, nearly forty years old, is one of inestimable value, both for scientific and practical purposes.

I have spoken of mental automatism as occurring *after* the paroxysm, but I must mention that according to some, I believe most, alienist physicians that degree of it which is called epileptic mania, although it usually occurs *after* a fit, does not always do so. It sometimes 'replaces' a fit. A patient who is subject to ordinary epileptic attacks may, on this hypothesis, have as it were *instead* a paroxysm of mania. There is what is called the *masked* epilepsy, described by Falret. It has been said that the patient who is subject to attacks in which there is convulsion of muscles, may at another time have an attack in which there is 'convulsion of ideas' and corresponding excess of external action (mania). I used to adopt the hypothesis of masked epilepsy. But I do

not now think it possible that a nervous discharge at all comparable in degree to that which causes convulsion would cause even such caricatures of normal action as occur either in epileptic mania or in slighter cases of mental automatism. I now think another hypothesis is preferable. I think it probable that there is a transitory epileptic paroxysm in every case of mental automatism occurring in epileptics before their mental automatism sets in. I am fully aware, and freely admit, that occasionally no signs of a prior fit are *discoverable.* The patient who at other times has ordinary convulsive seizures may become suddenly maniacal, although even when under observation he presented no physical change to indicate a paroxysm before the raving begins. To acknowledge this fully let me give an example. I wish to show that I look the difficulty full in the face. A very intelligent medical man came up to town with an epileptic patient of mine. He afterwards told me that the patient became suddenly, without any premonitory symptom to indicate an epileptic paroxysm, very much excited, struggling more or less violently with his attendants for twenty minutes. Nevertheless, I think it more probable (I think it a smaller hypothesis) that in this and in every case of sudden mental automatism in epileptics there has been a prior slight and transient paroxysm. I believe there is in such cases, during the paroxysm, an internal discharge too slight to cause obvious external effects, but strong enough to put out of use for a time more or less of the highest nervous centres. The mental automatism results I consider from over-action of lower nervous centres, because the highest or controlling centres have been thus put out of use. The automatism in these cases is not, I think, ever epileptic, but always post-epileptic. The condition after the paroxysm is duplex : (1) there is loss or defect of consciousness, and there is (2) mental automatism. In other words, there is (1) loss of control *permitting* (2) increased automatic action.[1]

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[1] I believe that there is a double condition in insanity, whether acute and temporary, as in epileptic mania, or chronic, as in insanity ordinarily so-called; there is a positive and a negative condition. I find that this opinion was stated long ago by Dr. Monro. The principle it illustrates was formulated by Laycock ten years earlier. The increased action (positive state, *i.e.* the raving, &c.) is owing

I cannot discuss this matter here; for medico-legal purposes it may, however, be assumed either that mental automatism always occurs after, or that in some cases it replaces an epileptic seizure. In most cases there is clear proof that it does occur after the seizure.

Scientifically, the slighter degrees of post-epileptic mental automatism are the more important, for these cases are simpler experiments on the organ of mind. Moreover, the study of them is desirable, as they give us a clue to the nature of the severer degrees. I use the word slight as the opposite of violent or severe, for cases in which there is great raving, &c. It is not enough borne in mind that the more imperfect and the shorter the paroxysm, the more likely is it that *elaborate* delirium and correspondingly *elaborate* automatic actions will follow. This is, however, what on Laycock's hypothesis of Reflex Cerebral Action and Spencer's hypothesis of Nervous Evolution, we should expect *à priori*. Hence the transitory slight seizure may be overlooked or not inquired for at all, and an opinion as to the nature of the case may be formed from one part of it, from the more persistent elaborate grotesque post-epileptic action.

Our first task in such an inquiry as this is to show that, what I have called elaborate, but what are better called highly compound actions, can be done automatically—*i.e.*, unconsciously. *It is not simply a question of the social impor-*

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what, metaphorically speaking, is loss of control of lower centres by the highest to contres, of which the function is lost or impaired (negative state, defect or loss of consciousness). This principle of over-action of lower centres as a consequonce of loss of control from inaction of higher centres was stated by Anstie in his 'Stimulants and Narcotics.' It was stated quite independently with regard to the epileptic paroxysm by Dr. Thompson Dickson. In this application of the principle I do not agree, but I think it applies to the very different condition of epileptic mania to which also Dr. Thompson Dickson applied it. See also Rutherford, 'Lancet,' April 29, 1871. My own hypothesis is, that the epileptic discharge 'removes control' by temporary paralysing the highest centres. The highest centres are temporarily exhausted, paralysed, or put out of use by the strong discharge, just as a subordinate nervous centre (corpus striatum ?) is by a discharge of convolutions near it in cases of hemiplegia after convulsions beginning unilaterally. (Epileptic Hemiplegia of Dr. Todd.) It must never be forgotten that in post-epileptic insanity the 'control' has been removed very suddenly. This fact bears, I think, on the interpretation of the violence which often characterises epileptic mania.

tance or violence of the actions, but of their degree of complexity.
This must not be forgotten. Here again I would urge the
importance of the slight fits. It seems to me to follow of
necessity that the slighter the fit the more complex the
mental automatism will be.[1] Again, it is not so much a
question of the enormity of the actions as of their absurdity.
On this view the recital of cases in which, after epileptic
paroxysms, actions in themselves trumpery occurred, is rele-
vant medico-legally as well as scientifically.

But even before speaking of slight automatic actions
developing after epileptic seizures, one may remark that if a
slight fit occurs while the patient is already employed in
something which is largely automatic, as, for example, playing
a well-practised tune, he may go on doing that automatic
thing—may continue playing correctly whilst unconscious.
The automatic action had, so to speak, possession of the mind,
and consciousness was not concerned in it before the paroxysm
occurred. Every one has seen a person play a simple well-
learned tune when talking of something else ; a transitory
lack of consciousness might not interfere with his perfor-
mance. In the following cases, however, *new* automatic
actions were developed, or those actions going on were
altered when the epileptic paroxysm had removed control.
It is very interesting, however, to note that the alteration is
sometimes not in the 'form' of the action, but in the 'con-
tents' of that form. (See cases on next page.)

The first few of the following cases read almost like un-

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[1] At least it follows on the principle of Dissolution, and on the supposition
that the discharge begins in the highest centres, as I believe it to do in the cases we
have here to do with where there is loss or defect of consciousness at or very soon
after the onset of the paroxysm. Of course I use the term Dissolution as the
opposite of Evolution. Nervous Evolution is from the general to the special, from
the simple to the complex. The highest nervous arrangements, the climax of the
Evolution, are the substrata of consciousness. The reverse process of Dissolution,
when it begins in these highest centres, will be from the most special to the
general, from the most complex to the simple, or (using terms which are in this
connection somewhat lax) from the most voluntary to the automatic. It is
obvious, then, that the shallower the Dissolution (the slighter the fit) the more
highly compounded will be the consequent mental automatism. For, the shallower
it is, the more are the nervous arrangements, next in speciality and complexity to
the very highest, spared.

scientific curiosities, but their indirect medico-legal interest is really very great indeed; their scientific value is also very great.

A patient who had consulted me for epilepsy, whilst standing taking leave turned of a leaden white and looked very ghastly for a short time. He swayed a little, but did not totter. After a moment he came to himself, but he did not remember [1] that he had just before this attack given me my fee. This instance is not much to the point, but it leads up to another. On the next consultation, after replying properly to a series of questions, he gave no answer to one. I waited a little time, and then, looking at him, I saw that he was grinning as if amused at something. Next, whilst sitting quietly in his chair, he tore a piece off a packet of prescriptions, and put it in his mouth. I took it away, but he picked up another piece from the floor and began to chew it. In about a minute more he came to himself, and then spat out into the fire a pellet of chewed paper.

The following is a note of the case of a patient under my care in the London Hospital, who had several degrees of seizures. He had little attacks (*le petit-mal*) and grave attacks (*le grand-mal*) of epilepsy. ' I was sitting on his bed taking his history, he sitting by my side holding the ink-stand. After asking him a question, and getting no answer, I looked at him. He remained sitting, but his head was a little drooped, and his face slightly pale. He still kept hold of the inkstand, and after a moment moved as if to put it down. I tried to get hold of it as it was tilting, but he pushed me away with the other hand. He was well again in about half a minute.' The fact that this man's mental auto-matism was on this occasion of no importance to himself or

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[1] On a minute scale this is an illustration of what is very often noticed, viz., that in failing memory recent events soonest fade away. Indeed to some extent this occurs, I submit, daily. Our very highest processes are 'swept clean' by sleep; there is a daily oblivescence. Probably the unconscious cerebration (reflex action of Laycock) is active during sleep. There are two halves of thought, tracing resemblances and noticing differences. The former is the more automatic. This is the duplex form of all thought whatever. I think that the first half, tracing resemblances, is that which is active in sleep. It is, I think, to exaggeration of this half of thought, the resuscitation of ideas one after another by organised resemblances, that delirium and insanity, &c., is owing.

to others, does not destroy its significance. Trivial as the affair was, his pushing me away shows most plainly an adjustment of actions done unconsciously, or, at any rate, with obscured consciousness, to external interference. We shall see the bearing of this later on.

Another epileptic patient of mine, when in an omnibus, blew his nose on a piece of paper, presumably after a slight fit. When he got out he gave the conductor 2*l*. 10*s*. instead of the usual coppers. This man was subject to both little and great seizures. It is important to note that the 'form' of these actions was correct.

A patient of mine was seized with a fit whilst feeling a gentleman's pulse; when he came round, in another room, he began to feel his sister's pulse, she being near him.

I have another patient under my care, who, his friends say, as soon as the fit starts runs out as fast as he can. He is also subject to temporary mental confusion, in which he is supposed to be intoxicated. On one occasion he took a fellow-workman's coat, and was accused of robbery.

In an earlier part of this article, page 111, I mention the case of a patient who became maniacal in a railway carriage. The following is an account of one of his slight attacks as recorded by a medical man who witnessed it :—

'Just after a walk, and whilst sitting down, K. had an attack in the garden. Head turned to the left; eyes the same. He was pale, and made three or four clucking noises; cheeks looked hollow and pale. He rubbed his nose, made odd grabs at his trousers, boots, &c.; moved his hands in a rubbing manner. Then, after sitting quiet, he looked round, examined a letter, and seemed to want to fix his whereabouts. I asked him the name of the town we were in. He did not know. Twenty minutes after he said he did not remember the attack, but remembered the incidents of the moment before.'

For some years this patient *invariably* looked at his watch immediately after each attack. He has not done this recently; but one day lately, some time after returning from the water-closet, he found the candle extinguisher in his waistcoat pocket. I suppose this was because 'he' had taken

the extinguisher for his watch during the loss of consciousness. The grabbings at his trousers, the taking out the watch, are evidently automatic actions : of course unconscious, and of course therefore irresponsible. It is, in one sense, grotesque to talk about such trumpery actions being irresponsible. But see how the consideration of such slight symptoms bears in this very case. Since all the above facts were written out, the patient became suddenly unruly, striking lamps in a place of public amusement, and afterwards in the street ; when going home, he struck his medical attendant ; next a boy who was passing, and also an interfering bystander. Now if this patient were suddenly and without provocation to injure some one seriously, we might show that he was really irresponsible, because we could bring evidence of former elaborate and yet non-criminal actions done unconsciously, and therefore irresponsibly, as well as evidence of the comparatively trivial outrages on public rights last mentioned.

The following case is the most valuable one I possess of post-epileptic mental automatism. Several years ago an educated man, 31 years of age, was under my care at the Hospital for the Epileptic and Paralysed for epileptic seizures—using, as I do, throughout this lecture, the term ' epileptic ' according to the accepted definition. He became unconscious, and bit his tongue in his severe fits, and slept several hours after each of these. He had had about sixty severe attacks. But he had also very frequently what he called slight ' seizures.' It is of these I wish to speak. These slight seizures were of different degrees. He used the words ' slight,' ' strongish,' and ' strong ' to describe his fits in the lists he supplied me with. After the slight attacks he did not sleep ; we may almost say that instead he dreamed only, or was somnambulistic. The following is a note written by him. The italics represent parts he underlined :—

' 20th. Unconscious ? for perhaps three-quarters of an hour, remember *ordering* dinner, but not *eating*, or paying for it, but did *both*, and returned to the office, where I *found myself* at my desk feeling rather confused, but not otherwise ill ; *was obliged to call at the dining-room to ask if I had been*

ill, and if I had had any dinner. The answer was *no* to the former, and *yes* to the latter question.'

At my request he again asked the landlady and waiters if he had been ill, but they had noticed no peculiarity. He ascertained, too, that ' My fellow-clerk, who usually goes to dine when I return to the office, says he did not observe any peculiarity, nor was I gone longer than usual.' The landlady told him ' that three weeks ago I paid for my dinner with half a sovereign, that she gave me the change, which I put into my pocket, and very soon afterwards I went to her and tendered a shilling in payment for the dinner, when she told me I had already paid for it, but that I did not appear to remember having done so.'

The following is the instance which makes the case most valuable. All the details are important :—

' My wife and her sister being present, had been talking about supper, when it was agreed that my wife and I should have some cold fowl, and the sister some cocoa if there were any fire. She went into the kitchen to see, and reported that there was one. Soon after I began to feel chilly after being so warm with gardening, and I said I would go down to the fire. I did so ; and after standing there a few minutes, I felt symptoms of an attack, and sat down, I believe, on a chair against the wall. And here my recollection failed, the next thing I was conscious of being the presence of my brother and mother (who had been sent for as they lived opposite), and I have since been informed by my sister-in-law that she came into the kitchen, and found me standing by the table mixing *cocoa* in a dirty gallipot, half filled with bread and milk intended for the cat, and stirring the mixture with a mustard-spoon, *which I must have gone to the cupboard to obtain.*

'This caused them to send for my friends, to whom I talked, showing no surprise that they were there, and entirely unconscious of what I had been doing until told this morning. After I had recovered, I partook of some bread and butter, and a glass of brandy and water. I went to bed, where I passed a good night, though I woke early and could not go to sleep again.'

The bearing of this case is that if the automatism in-

stead of being a caricature of innocent normal actions, had been ' criminal ' and equally elaborate, the patient would have had a bad chance of escaping punishment. As the facts stated under date of 20th, page 116, show, he had fits which were too slight to attract attention ; the case indeed illustrates what was said (p. 113), that the slighter the fit the more complex the automatism after it. What form a man's mental automatism will take depends, I think, very much on what his natural disposition is. A savage and suspicious man would, when a fit had temporarily removed his highest faculties, more likely have killed some one than have mixed cocoa. And just as this poor fellow went to the cupboard for a mustard-spoon, so, had he been a savage man, he might have gone to another room for a poker when his sister-in-law came in. The value of the case is that it shows very elaborate actions done unconsciously. We must not under-rate the mental automatism, because what was done, not being violent, was unimportant. I repeat, the significant thing is the *complexity* of the action. Had an equally elaborate action ended in murder, it would have looked intentional. True, it would have been equally *absurd* had he killed his sister-in-law ; but when emotions are strongly excited, most of us are not logical. If a man ·or woman killed a beloved child, we, as scientific men, have to do with the question of absurdity or insanity of that action rather than with its enormity. It might easily have happened that the prior suggestion had not been about supper and cocoa, but about burglary. He might have been reading or hearing accounts of robbery which should be justifiably resisted. He might have automatically killed his sister-in-law instead of automatically mixed cocoa for her in a dirty gallipot.

Let me at this juncture mention a case in which also there was proof of external suggestion modifying post-epileptic automatism. One of my epileptic patients would, after an attack of epilepsy, get up unconscious and go about as if looking for something, or he would walk about unconsciously with his tools in his hands. Such things are, however, not much to the point ; they are very common. But it was said that he would talk after a fit of what he had been reading. Thus, one day he had been reading in the newspapers of the

Queen's way of bringing up her children; after his next fit he said that the 'children were all put under the Queen's shawl, and were going up above'—a grotesque and childishly-poetical statement of maternal solicitude, and its recompense if 'above' meant heaven.[1] We shall again have to speak of the influence of external suggestion before the paroxysm itself, or indeed occurring in the paroxysm,[2] and before the mental automatism sets in. I would now add to the statement that the slighter the fit the more highly compounded the actions permitted, the statement, 'and the more are they developed by external circumstances occurring just before, during, or after the paroxysm.'

I now state cases with a criminal aspect, but essentially similar scientifically, in order to illustrate how the trivial cases bear. I begin with trifling crimes. I had a boy under my care who was subject to fits, beginning by a 'subjective' sensation of smell; he would turn pale.[3] To use his mother's expression, he would often after the seizures 'go right out of his mind.' He was a shoe-black; and once after a seizure threw his blacking-box at a policeman. This was the 'crime.' For this he was taken before a magistrate, and fined five shillings. One evening after a fit he got into a rage, and said that a gentleman in the street had offered him five shillings to clean his boots. Later, he was taken to a lunatic asylum.

The following is an account written by an epileptic patient's mother. She begins by speaking of her son's fits. 'When I first observed them he became suddenly senseless, and remained in the same position as when he was first seized.

[1] An aphasic patient under my observation made many mistakes in speaking; he made one which reminds me of the epileptic's observation mentioned in the text. He said, 'Where is your little chapel?' instead of 'Where is your prayer-book?'

[2] By external suggestion 'occurring in the paroxysm' I allude to developments of sensations of colour, smell, at the outset of the seizures, &c., and to hurts received in falling; all are practically external.

[3] It was an interesting and important fact that he would hold his nose when the fit began. No doubt as his consciousness became obscured 'he' believed the smell was of something actually outside. It is, perhaps, too strong an expression to say '*he* believed,' for where is the '*ego*' when a man is unconscious or partly so? We may say that the smell developed an epileptic dream. I had a woman under my care who at the beginning of an attack had 'subjective' sensation of smell, who said, 'What a dreadful stink there is in the place!' She believed the smell was of something in the room.

Sometimes, if he had an attack when standing, I have known him fall on his knees ; also he has had fits in the night, when he foamed at the mouth and bit his tongue. [He had *le petit-mal* and *le grand-mal*]. On one occasion he had a fit at a place of worship during the sermon and took off his coat, sitting like that till he recovered his senses. At another time he was taking his tea alone when my servant heard he front door opened and shut. He had left the house without his hat, and was away about three-quarters of an hour. When he recovered himself, he found he was walking round the church near our house. This occurred at night. On many occasions he has gone out in a similar way, and *he invariably now rises and unbuttons his waistcoat and trousers.* Last Sunday he had a fit at another place of worship, and took off his boots. He has had many in the street. His memory is always very bad for some time after an attack.'

Here is an account of another attack written by the patient himself :—

' I was in a shop at Twickenham seeking an order for wines ; I suddenly fell on my back—the first time I have allen in a fit for six or nine months. The shopman at once cae from be hind the counter, and with the assistance of a iend r aised me. I instantly threw my arms about and shook both off, but they caught hold of me again and led me out of the shop, when the friend left and I again made my escape from the shopman, leaving my hat and order-book behind. The shopman then got a man to mind his shop, and he and two policemen went in search of me, all going different ways.

' He first discovered me a quarter of a mile away, *asking for my hat at all the shops, but not having recovered my senses, nor did I until I got to the railway ten minutes after.'*

That he asked for his hat at the shops before he gained consciousness is important, as showing normal actions done unconsciously. Had he committed a ' crime ' when apparently so much himself, but when really so unconscious, one would have little hope of convincing a magistrate of the poor fellow's irresponsibility.

On one occasion he unbuttoned his trousers when there were four women present (see the part of his mother's letter

italicised). It is quite clear that this poor fellow will run great risk of being indicted for an indecent offence. Fortunately the four women were members of his own family. It would be hard to convince a magistrate or a jury that he was not fully aware of what he was doing if he unbuttoned his trousers in the park under certain circumstances. It is possible that this action arises from the notion that he has to make water, and not from any sensual excitement. His condition after a fit to be next mentioned might, I imagine, have been easily altered into a quasi-criminal one had he been roughly interfered with, or had he been naturally of a savage disposition. I gathered the following from him at a visit to the hospital. I copy from my case-book :—

' A few days ago, he went into the back parlour as he felt he was going to have a fit. His mother followed him, and found that he had taken a knife out of his pocket and was grasping it, not by the handle, but by the blade. His mother took it away, shut it up, and put it behind the bookcase. He went to the bookcase, got it again, and then kept waving it about, but it was now closed. His mother then got assistance, and the knife was again taken from him.'

His taking the knife and his getting it again when it had been taken from him, are medico-legally very interesting facts, showing eborateness ; on the other hand, taking the knife by the blade was absurd.

As some evidence of this patient's general competency, I may say that his father threatens to turn him out of doors for not supporting himself. He is an intelligent man, seems very calm and sober-minded, and, in spite of what his mother says, appears to be in good general health. In the intervals of his fits his memory is impaired, but not to any great extent. Yet the fact that he is occasionally for a short time incompetent from an attack, renders him unable to keep his situations. He has been a traveller for orders, and when walking he ' loses himself,' but goes on walking. In one fit he lost himself at Blackfriars, and came round again when at the Elephant and Castle. He has been knocked down by an omnibus, and has once nearly walked into the Thames. He remarks, and this is worthy of consideration, that he is quite safe in these walking attacks if he is alone. This, I

think, supports the speculation that if roughly interfered with in the fit in which he was waving the knife about, his doings might have altered to graver actions.

I now pass to speak of cases in which the patient's doings were violent, a separation which is arbitrary, although convenient. The violence and outrageousness of the seizure depend doubtless very much on the natural disposition of the patient who suffers from the attack. There was no criminal action in the case I relate, but the 'material' for crime was abundant. This case is of a very common kind, and therefore I need the less regret that my notes of the non-mental part of it are meagre.

A man, 47 years of age, had been subject to severe fits for seven or eight years when I first saw him. At first he used to sleep after his fit; but after the first few 'he raved instead.' This was his own expression, and is no doubt essentially correct. He dreamed instead of sleeping deeply, as is most common after fits, and his raving was the external sign of his dream. After a fit at the hospital he was asleep or comatose for awhile before he raved. The order of events in the paroxysms was this—(1) coloured vision; (2) convulsion (sometimes, I was told, of one side, sometimes of the other) and insensibility; and (3) after the paroxysm raving. He had a fit at the hospital one day. I was called to him. He was out of it when I saw him, was very dark, and breathing hard as if half suffocated. I left him after awhile, but was called again soon, and found him up shouting, looking *alarmed*, and yet apparently lost to what was about him ; he struggled with us, but did not look at us. His fears were evidently of something else, of something in 'a dream,' no doubt. This state of things lasted about a quarter of an hour. He then seemed simply stupefied and despondent, but could answer simple questions.

As he seemed an intelligent man, I asked him to write out an account of his coloured vision, for indeed this is the part of the case which then interested me most.[1] The words

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[1] The coloured vision was the subjective side of what was objectively the beginning of the cerebral discharge, which, increasing in strength, and therefore spreading, led to convulsion. Sometimes the discharge does not increase in strength, and does not spread. Sometimes it did not in this case ; the patient had occasionally attacks of coloured vision only. Similarly a man subject to fits

in square brackets are replies to questions I put to him after reading what he had written, or observations of my own :—

'Before having a fit I have a throbbing light of various colours [green, yellow, and red], in the right eye, which lasts sometimes 15, 20, or 30 minutes, which takes my sight and senses away, and then I go off into a fit, making a loud noise as if I were frightened. I then lie very quiet. I am much convulsed, sometimes for 20, 30, 40 minutes [no doubt a succession of fits], and when I am coming to out of the fit, I rave like a maniac, and gradually come to my senses in about half an hour [of course this is what he was told]. I sometimes have those throbbing colours in my right eye, which last a few minutes and stop suddenly, and then I do not have a fit, only feel giddy for a short time. I mostly have a fit about every four or five weeks.

'Saw the doctor on Monday, February 16. The next day (Tuesday, 17th), had pains at back of ears; had those colours in my eye, which lasted about five minutes and stopped suddenly; felt as if a fit was coming on.

'February 26. About ten o'clock in the morning was taken very bad with the throbbing in the right eye; could see various colours floating about, and lasted so until twelve. It then went off into a fit which lasted about an hour, was very much convulsed, came to a bit, and raved very much; got gradually better and got my senses about three o'clock; my eyes been very much bad with throbbing, and bad-sighted since.'

This patient's case has only an indirect medico-legal interest; I mean, it is for this purpose too simple, as the man was *known* to be subject to severe epileptic fits. But were the

beginning in his index finger and thumb may have sometimes twitching of those digits only, and sometimes this twitching will lead on to a one-sided, or even further, to a universal convulsion.

I believe that this patient's seeming alarm in the post-epileptic raving was the result of some terrible dream developed in him by the coloured vision. I believe that the colour first developed in epileptics, when colour is the first symptom (warning or aura), is usually red; it is not always so. I find that this is not a novel observation. Falret has pointed out that a premonitory symptom or beginning of an epileptic seizure is often red vision, but he adds ' or purple.' Again Falret, speaking of epileptic maniacs, says, 'They constantly see luminous objects, flames, circles of fire; and what is worthy of *remark, the colour red or the sight of blood* frequently predominates in their visions.'

man to have killed some one in his first maniacal attack, the prior paroxysm not having been witnessed, he would possibly have been hanged.

We have sometimes in the same patient post-epileptic doings of different degrees, from a sort of quiet somnambulism to violent struggling. Seven years ago I had under my care a boy, 19 years of age, who had abnormal mental states after epileptic seizures, which illustrate this remark. The following is an account his father gave me. The exact onset of this condition was not noted in the symptoms to be first described, but in other seizures his father said that 'the lad's eyes are open, he gets stiff, grinds his teeth ; the next [no doubt post-epileptic] is trying to get away, struggling,' &c.

Last time he had a fit and went to bed, and when in bed said, ' Wait a bit, Bill. I am coming.' He went downstairs, he unbolted the doors, and went out in his night-shirt. He came to himself just as he was stepping on cold stones, and then his father touched him. He said that he had had a dream : ' It's all right, I have had a dream.' He went to bed, and had not been in bed five minutes when he began again talking of Bill (an acquaintance in the volunteers), saying, ' You are in a great hurry to get your coat on.' His father went into his bedroom again, called his brother, and got the patient into bed. Then a fit, so his father says, began (I suppose it to have been post-epileptic automatism), and they were obliged to get a policeman. The policeman, I was told, got on the patient's chest, the others got hold of his arms and legs. The struggling only lasted about three or four minutes, but returned many times during four hours. This struggling was evidently mania.

The following case is the one of most direct medico-legal interest. I have to thank Mr. Rivington for permission to use it. It was a case of self-mutilation under his surgical care, in January 1874. I have to thank Mr. Mercier for taking notes of it for me. I beg attention to all the details Mr. Mercier gives ; they are all important, either medically or legally. Any imperfection in the report is, I think, owing to difficulties in getting information. For any imperfections otherwise caused, I am at least fully responsible, as I frequently saw the patient with Mr. Mercier. I read his notes carefully

from time to time. In January 1875, Mr. Mercier called on the patient, and obtained clearer accounts of some things in the case; but it will be well to give the report, showing how the case appeared when he was admitted January, 1874— a year before. It is most important to remark that many of our hospital patients or their friends, as do many of our private patients, give imperfect, erroneous, or confused accounts of their former conditions.

Family history.—A woman, aged about 35. Patient's maternal grandmother was in a lunatic asylum. Her maternal uncles and aunts died young. Her mother was melancholy, taciturn, and suspicious, and used frequently to say that she had 'hosts of enemies.' There is no history of cerebral disorder on the father's side. The patient's children are all healthy in mind and body; the second died of convulsions.

Personal history.—The patient herself has never exhibited any signs of madness before the present outbreak, but she has always been irritable and passionate. She never did odd things. (*Vide infra.*)

Patient's own account.—She was married thirteen years ago. Before her marriage she used now and then to faint away; she would fall down and lose consciousness, but never had convulsions. About a year after her marriage she began to have 'fits,' attended by loss of consciousness and convulsions. When the fit came on she would fall suddenly and remain unconscious for an hour, more or less. These fits recurred at intervals of about a month, and came on chiefly at the menstrual periods. She also had them during pregnancy.

History of present illness.—On the afternoon of January 19, 1874, the patient was cutting bread for her children's tea, when she suddenly sent them all out of the room, and was found a short time after lying in a pool of blood with a deep gash in the left arm, which divided all the structures in front of the elbow, and laid the joint open. It was then found that she was out of her senses; she was at once brought to the hospital, and was then in the following condition.

Patient's state on admission.—She had evidently lost a great deal of blood; her face was extremely pale, her lips livid, her radial pulse at first wholly inappreciable. Her face

had a most peculiar, wild, horror-struck expression, due to her eyebrows being raised, and the upper eyelids lifted quite away from the upper edge of the cornea; to her nostrils being much dilated, and her lips drawn in. She was excited, maniacal; she tossed her arms about, tried to get off the stretcher, asked for her husband, who had been dead some months, confused the people around with her acquaintances. She seemed to feel no pain from her arm, although she occasionally looked at the wound and said it was cruel. *She accused different people of doing it.*

Gradual subsidence of mania.—Her wound was dressed, and she was put to bed, where she soon became much calmer, and the peculiar expression before noticed disappeared from her face. Several times, however, it suddenly returned, and she struggled to get out of bed. She asked for water once or twice, but except this uttered no rational sentence. During the night she slept for several hours.

Return of sanity on the morning after—Relapse at night.— January 20, second day. She appeared utterly different, her face was flushed, her lips natural red, her face was composed, and she was conscious and rational. She remained quiet and sane throughout the whole of the day, but at night was raving violently, trying to get out of bed and do herself an injury.

Variations in mental state.—For the next two days the patient remained quiet, conscious, and rational. On the morning of the fifth day she appeared uneasy; she looked anxious, and said that she was surrounded by enemies. That night she was raving and furious; it took two people to hold her down. All her violence, however, was directed against herself. The next morning she explained that she had seen things about her; when asked what things, she said, 'Wolves.' During the sixth and seventh days she was quiet and rational, but had a remarkable expression of face; she looked half frightened and half suspicious, as if she anticipated harm. In the evening of the seventh day I was told that she was wild again. I found her lying in bed, not struggling, but crying out, 'The world is coming to an end, cut my throat, let me choke myself;' and this she endeavoured to do. On speaking to her firmly and peremptorily, she immediately

answered rationally, and replied to numerous simple questions quite correctly. (Such questions as the day of the week and month, the ages of her children, &c.) She had then been raving about a quarter of an hour. She remained quiet for about five minutes, and then began again in exactly the same style, saying that the world was at an end, and that we were all at the bottom of the sea, and desiring to have her throat cut. When she is very excited she will not answer a question, but when only moderately so she will break off from exclaiming that the world is at an end, to reply to a simple definite question, or to perform any action that she may be told to— *e.g.*, to shut her eyes, put her tongue out, &c. She would even in reply to questions tell a definite connected story about her children. The next moment she was raving as before. Her tongue was at this time strongly deflected to the left. There was no convulsive action of any part before the attack. Ten minutes later she was quiet and rational.

Subsequent history.—Since that time she has had no outbreak of such violence; but she often expresses the utmost anxiety about her children, fancying that they are secreted somewhere about the hospital; or about herself, fearing that she will be sent away or not allowed to go, or some other equally reasonless fear. She sometimes has visual, but does not have auditory spectra.

The wound healed well, and the patient got into apparent good health, mental and physical.

A year later, January 10, 1875.—Emily A—— has had very good health since she left the hospital. She has had numerous 'fainting fits,' in which she loses her senses for a few minutes, but none of the long spells of unconsciousness that she used to have. She had always contrived that no one should see her in the fits, so that no particulars were to be obtained from others, but she herself says that she never does odd or strange things after the fits now. She says, however, that before she entered the hospital she used frequently after the fits to do very strange things. She would 'turn the house upside down,' and 'pull the beds to pieces,' and then wonder afterwards who could have been doing it. She has never harmed herself or any one else, except on the one occasion when she came to the hospital.

With regard to that fit she states that she felt very bad all the morning, and that she went to a doctor's in consequence. In the afternoon she felt so bad that she had to lie down; and shortly after, when her son came and asked her for some bread and butter, she went downstairs, took a knife out of the drawer, and then feeling very ill came and lay down again without cutting the bread.

After this, she remembers no more until she was brought to the hospital.

Present state of her arm and hand.—The muscles on the palmar aspect of the fore-arm are atrophied, as are the thenar muscles. The interossei do not look wasted. The thumb and two outer fingers are livid, cold, and the skin is smooth and shining, unlike the others, which look normal. The nails of those three digits also are distorted.

The position of the two inner fingers is normal. The proximal phalanx of the two outer is extended beyond a right line with the dorsum, and the two distal phalanges are flexed. The thumb lies along the index and she cannot extend it. The wrist is strongly flexed and cannot be extended, owing to contraction of flexor muscles.

Sensation on thumb and two inner fingers dulled but not lost, and localisation is imperfect.

She is stout, florid, and looks in good general health; does hard work.

That this was a case of mental disorder following an epileptic seizure, was, I think, practically certain. She had had slight attacks, faints, &c., which were no doubt *petit-mal*, and also convulsive seizures. I should believe that she had had a slight attack of *petit-mal* before she sent her children out of the room, for it is to be repeated that elaborate mental symptoms more commonly attend those cases of epilepsy in which there are little attacks (*le petit-mal*). In Mr. Mercier's second account (1875), *vide* page 127, there is a history of strange doings after attacks ('pulling beds to pieces,' &c.) The suddenness and the grotesque absurdity of the action are complementary evidence. The action was more important than the cocoa-mixing (see p. 117), but scientifically it was about on a par with it—that is to say, it was

about an equally highly compounded action. It also was developed by external circumstances; the knife was in her hand, for she was about to cut bread when taken ill.

Self-mutilation is itself a crime. It is no great stretch of imagination to assume that it was a sort of accident that the woman did not cut the throats of her children instead of her own arm. A case thus presented would not be easy to the laity. It was not known that she had had fits. It was the woman's first outbreak of mental disorder, or rather the first striking attack; this would have told against her. Her case well illustrates the importance of studying grotesque actions as well as violent actions. There was seeming method in what she did. She sent the children out of the room. This is analogous to the act of the cocoa-mixer, page 117, who went to the cupboard for a mustard-spoon. It might have fared badly with her had she sent out an elder son and cut the throat of a little child. Moreover, her subsequent raving, although genuine, was of a common, wild, very incoherent sort, and might have looked like pretence to a suspiciously minded layman's 'common sense.'

I have already given evidence from the mental automatism of simple cases, that elaborate and highly compound actions may be gone through when a patient is unconscious—presumably after an epileptic seizure. I have also shown that the mental automatism may be developed by suggestion, or altered thereby. She was cutting bread when, as I suppose, the fit (or, as she would say, 'faint') took her.

In a legal inquiry, the facts as to heredity are exceedingly important, but their bearing is so obvious that I should be wasting time were I to dwell on their importance in such a case. The *nature* of the insanity in the mother must be mentioned; she was suspicious, and had, she thought, 'hosts of enemies.' The patient herself was irritable and passionate. Her accusation of others when she came to the hospital is noteworthy. Her mental condition at that time might easily have led people to suppose that she had been injured by some one else, except indeed for the nature of the injury. Then her subsequent mental condition was significant—she was 'surrounded by enemies.'

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ON THE APPEARANCE OF THE RETINA AND CHOROID DURING THE ADMINISTRATION OF CERTAIN DRUGS.

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The circulation of blood in the fundus oculi has of late years been considered so intimately connected with the cerebral circulation, that the condition of the brain in that respect may be estimated by the corresponding appearances of the optic disc, retina, and choroid. Consequently, as certain forms of brain disease are supposed to be associated with hyperæmic or anæmic conditions of the brain, it has been anticipated that therapeutic agents, administered with the intention of affecting the blood supply or nutrition of the brain, may reveal their action by the circulation within the eyes.

The following experiments were made with the view of ascertaining whether the circulation within a normal eye is affected by drugs.

Rabbits were used for observation, because in their eyes the nerve-fibres after entering at the disc still retain their neurilemma in the retina for a considerable distance, spreading forwards and backwards in two broad fan-like tracts, and give a bright white background to the blood-vessels which course over them, so that the very smallest branches are seen, and if any change takes place either in their colour, shape, or size it is readily detected.

The hand ophthalmoscope was discarded, as prolonged observation cannot be conveniently kept up with it, and the slightest change in focus by increase or diminution of dis-

tance between the mirror lens and eye alters the appearance of the vessels in hue and size, so the comparison at different times cannot be depended on.

The rabbit was always securely fixed on Czermac's rest with the side of the head looking slightly upwards, to bring the optic disc directly into view, and the eyelids were kept widely apart with a spring speculum, which also held back the membrana nictitans.

Mr. Priestley Smith's demonstrating ophthalmoscope was used, and after focussing the instrument correctly in every case, the eye was kept under observation for any desired length of time.

A rabbit was fixed in the rest with the eyelids kept widely apart, and the arteries and veins of the retina were observed for hours; the disc and choroid were also easily seen at the same time, but no change whatever took place from the constant glare of reflected gaslight from the mirror, nor was the slightest change produced by the instillation of a few drops of a solution of sulphate of atropia into the eye, so as to dilate the pupil to its widest extent. Since atropine was found to have no effect on the circulation when applied topically to the eyeball, it was generally used to dilate the pupil, after the eye had first been observed without it. When the eyelids are kept widely apart by the speculum, the cornea, on prolonged observation, is apt to get less transparent from a deficiency of lachrymal secretion flowing over it; but it is always quickly restored to its usual condition by syringing a few drops of water against its surface, when the fundus of the eye is as clearly seen as at first. The pupil usually contracts when the light is first thrown into the eye, but in a few minutes it dilates again, and the iris remains at rest. In all cases the number of the retinal vessels seen with their branches was counted, and in most pencil sketches of their distribution were made, for reference while the animal was under observation and to compare with the appearances on dissection of the eyes after death. The curves in the course of special branches were taken note of, and the animals were always kept under observation on the rest for more than half an hour before any drug was ex-

hibited, so that the characters of individual branches might become familiar.

Rabbits were several times put under the influence of chloroform and ether by inhalation, and hypodermic injection to profound insensibility, without affecting the circulation of the retina or choroid in any way. Great attention was paid to the choroidal vessels in the eyes of albino rabbits, with the same negative result. So the toxic agents were usually administered till the animals died.

NICOTINE.

At 2.0 o'clock P.M. Respirations 116 per minute and pulse 252. The rabbit's ears were cool and pale.

At 2.23. Half a minim of nicotine was injected[1] under the skin, and within a minute after it was convulsed, breathed stertorously, and was hypersensitive to touch. Its ears kept cool.

At 2.29. It had a severe struggle.

At 2.30. Respirations 90, pulse 240. Pupil of eye under observation contracting.

At 2.35. Struggled severely and was convulsed all over; the muscles of thighs kept twitching for some minutes. The vessels of its ears got gradually fuller of blood.

At 2.40. Respirations 180, pulse 320. Heart irregular in its action and pulsations can with difficulty be counted. Ears warmer and more injected with blood.

At 2.45. Half a minim more of nicotine was injected under skin.

At 2.50. Respirations 152, pulse 172. Action of heart weak and irregular, at one time pulse 320. Has a convulsion occasionally. Insensible to pricking.

At 3.0. Ears hot and keep injected with blood. Left leg and all the body affected with clonic spasms, but the right leg kept at rest.

At 3.3. The right side of body, right leg, and right ear now affected by jerking spasms.

At 3.5. Respiration ceased. Both pupils contracted firmly.

No change whatever took place in the appearance of the vessels of the retina or choroid during the whole time of observation while the rabbit was alive.

At 3.10. The arteries and veins of retina were in a slight degree smaller in calibre than when the animal was living. There was no part lost to view nor empty of any of the blood-vessels, neither was there any beading nor break in the column of blood in the vessels.

No difference could be detected in the choroidal glow. Both pupils kept contracted, and the ears remained partly injected.

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[1] Concentrated solutions were used, and when injections were repeated they were always made in fresh places of the skin.

In the left eye one of the retinal arteries, as it curved over the margin of the disc, contained at that part only so much blood as to make it discernible, the rest of its course and all the other vessels appeared as in the right eye.

After death, when the rabbit was removed from the rest, some urine escaped from its bladder.

Autopsy at 9.0 P.M. :

Brain : The sinuses of the dura mater contained a quantity of dark fluid blood, and the pia mater was very vascular.

Eyes : A quantity of dark fluid blood escaped from both orbits when the eyeballs were being enucleated. The vessels of both eyes were in exactly the same state as seen with the ophthalmoscope after the death of the rabbit.

Heart : Left ventricle was contracted and empty. Right ventricle was full of dark clotted blood. Right and left auricles were full of dark fluid blood.

The lungs, kidneys, and liver were normal.

The bladder was almost full.

Rigor mortis was present.

ATROPINE.

At 3.30 P.M. Its ears were hot and the blood-vessels full.

Respirations 100 per minute. Pulsations of heart too quick to be counted ; it was very excited. The pupils were at rest.

At 3.32. One-fifth of a grain of sulphate of atropia in concentrated aqueous solution was injected under the skin, and in two minutes after the pupils were widely dilated.

From the beginning a large vein and artery, with nine primary and other secondary branches, were kept under observation.

At 3.43. Respirations 88, pulse 240.

At 3.45. Two-fifths of a grain more of sulphate of atropia injected under the skin.

At 4.20. Respirations 52, pulse about 300—almost uncountable, so quick. Breathing abdominal.

At 4.25. Three-fifths of a grain more injected.

Respirations 44. Cardiac pulsations could not be felt. It was insensible to pinching.

Its ears were extremely injected.

At 4.40. Respirations 45, pulse 304—counted with difficulty, as the pulsations of heart were very small and rapid.

At 4.45. Three-fifths of a grain more injected. Ears kept hot and injected.

At 4.55. Respirations 44, pulse 320.

At 4.58. Three-fifths of a grain more injected. It gave a short, weak, convulsive struggle every quarter of an hour or so, and was quite insensible.

At 5.5. Respirations 42. Pulsations of heart could not be felt.

Ears hot and extremely injected.

At 5.10. Three-fifths of a grain more injected.

At 5.15. Respirations 43.

At 5.20. Three-fifths of a grain more injected.

At 5.23. Respirations 44. Mucous rales in trachea, and was breathing stertorously.

At 5.30. Respirations 44.

At 5.35. One and two-fifths grains more injected.

At 5.50. Respirations 39.

At 6.50. Dead. Pupils were dilated. The blood-vessels in the right ear were very full, in the left not so full.

During life no change whatever appeared in the circulation of the retina and choroid; while after death the blood-vessels of retina retained their calibre, but in some parts of their course contained less blood than when the animal was alive.

Autopsy at 2.0 o'clock on the following day : Rigor mortis was present. Both ears were very pale.

The pupils were at rest—neither dilated nor contracted.

The lungs, kidneys, and liver were normal.

The brain and its membranes were normal.

Heart : The left ventricle was firmly contracted and contained a very small dark clot. Right ventricle was full of dark and soft clotted blood.

The right and left auricles were full of dark clotted blood.

The bladder was full.

The blood-vessels of retina were easily recognised, and appeared as when last seen with the ophthalmoscope.

ACONITIA.

At 3.0 P.M. Respirations 64, pulse 308—quick from excitement.

Its ears were cool, and the central vessels of the ears were empty.

At 3.20. One-fifth of a grain of aconitia was injected under the skin.

At 3.23. Respirations 42, pulse 376.

Almost insensible to pricking.

At 3.30. Respirations 62, pulse 336.

Ears kept cool and not injected. The heart's impulse was better felt.

At 3.40. Two-fifths of a grain more injected.

At 3.45. Respirations 46, pulse 360.

At 3.55. The saliva began to drop from its mouth.

At 4.0. Respirations 22, pulse about 320 and weak.

Ears not more injected than usual.

At 4.5. One-fifth of a grain more injected.

Its ears kept cool and their circulation quiet.

No change took place in the fundus oculi.

At 4.10. Respirations 17, pulse 280, weak.

At 4.20. Eight-fifths of a grain more injected.

At 4.25. Respirations 34, pulse 280. Is still salivated.

At 4.45. Salivation ceased.

Four grains more injected.

At 5.0. Breathing laboured.

At 5.20. Dead.

During life no change took place in the vessels of the retina or choroid. And after death the colour of the choroid remained the same. Only one vein in a short portion of its course near the disc appeared attenuated in a very slight degree; all the other blood-vessels of the retina remained the same as when the animal was alive. There was no diminution in the quantity, nor break in the columns of blood in the vessels; nor were any of the finest branches of the vessels that had been observed during life lost to view when the animal was dead for some time.

Both pupils at death were dilated and remained so.

Autopsy at 3.30 P.M. on the following day: Rigor mortis was present. The pupils of both eyes were widely dilated.

The brain and its membranes were not congested; there was fluid blood in the sinuses of the dura mater.

On removing the eyes some fluid blood escaped from the orbits.

The veins of the retina contained more blood than the arteries. All the branches were seen that had been under observation, and the apparent narrowing of the vein, as seen with the ophthalmoscope, in a part of its course, still existed.

Heart: Left ventricle was partly contracted and contained a little black clotted blood.

Right ventricle was full of black soft clotted blood.

The right and left auricles contained black clotted blood.

HYDRATE OF CHLORAL.

At 8.0 P.M. Respirations 123 per minute, pulse 280.

Twenty grains of chloral hydrate were injected under skin.

At 8.5. Respirations 136, pulse 312.

Ears were cool and pale.

At 8.18. Respirations 122, pulse 320.

When the rabbit's tail was pinched, the peculiar jerky cry during expiration was elicited.

At 8.20. Twenty grains more was injected.

At 8.25. Respirations 94, pulse 336.

At 8.33. Respirations 88, pulse 344.

At 8.37. Twenty grains more injected.

The eyeball rolled downwards slowly now and again.

At 8.42. Respirations 72, pulse 320, but so weak as to be counted with difficulty.

At 8.50. Respirations 88. Heart's action was irregular and weak, so that the pulsations could not be counted.

At 8.52. Twenty grains more injected.

At 9.0. Respirations 88.

At 9.8. Forty grains more injected.

At 9.16. Respirations 70.

Was in a state of deep coma, and could not be roused.

Its ears kept cool and pale.

At 9.28. Respirations 52.

The pupils slowly contracted. A few more drops of atropine were instilled into the eye, but had no effect on the iris.

It did not give the cry when pinched.

At 9.36. Respirations 47.

At 10.0. Respirations 31.

No change whatever took place in the vessels of the retina or choroid.

At 10.3. Died.

The pupils got more dilated after death, and the large blood-vessels of the retina got perceptibly smaller, but the branches were all seen, and no break took place in the columns of blood.

The choroid did not look any paler, but retained its red glow.

Autopsy at 8.30 P.M. the following day: Rigor mortis present.

The blood-vessels of the dura mater and pia mater were moderately full. The brain looked neither pale nor congested.

The lungs were congested.

Heart : The left ventricle contained a small black clot. The right ventricle was distended with dark fluid and clotted blood.

The right and left auricles also contained dark fluid and clotted blood, but were not much distended.

The kidneys and liver were normal.

The bladder contained urine.

NITRITE OF AMYL.

At 7.40 P.M. Respirations 64 per minute, pulse 280 per minute.

Its ears were cool and pale.

At 7.50. Twenty minims of nitrite of amyl were injected under the skin, and in three minutes after the rabbit's ears got hot and intensely injected with blood.

At 7.57. Respirations 54, pulse 336.

At 8.0. The rabbit was quite insensible to pinching.

At 8.5. Respirations 56, pulse 340.

The ears were still intensely injected, but no change whatever took place in the circulation of the retina or choroid.

At 8.10. Respirations 80. The pulse was quick and irregular, and could not be counted.

The ears still kept hot and injected.

At 8.15. Respirations 88.

At 8.20. Twenty minims more were injected.

At 8.22. Respirations 100. The rabbit struggled convulsively and defecated.

At 8.25. Respirations 98.

At 8.30. Twenty minims more were injected.

At 8.32. Respirations 118.

At 8.35. Forty minims more were injected.

At 8.45. Respirations 60. Pulsations of heart could not be felt.

The ears got paler. The pupils of both eyes got extremely dilated.

At 8.50. Respirations 32, and of a gasping character.

The ears were pale and cold.

The respirations got gradually slower.

At 8.55. Dead.

No change took place in the vessels of the retina or choroid during life, but after death the vessels got gradually smaller.

Half an hour after death ouly the very smallest branches were lost to view, through being quite empty. There was no break in the columns of blood that remained in the larger vessels, and they did not get empty.

The disc was pale.

The choroid still retained the red glow, but not so deep as when the animal was alive.

Both pupils ere widelwy dilated.

When the rabbit was removed from the rest after death, urine escaped from its bladder.

Autopsy at 11.0 A.M. on the following day: Rigor mortis was present. Both pupils were somewhat dilated, and the ears were pale.

Brain: The sinuses of the dura mater did not contain much blood. The blood-vessels of the pia mater were very numerous and distinct, though not greatly distended with blood. No blood escaped from the orbits when the eyeballs were removed.

Heart: The left ventricle was contracted and empty. The right ventricle and auricle were disteuded with dark fluid blood.

The left auricle contained black clotted blood.

The lungs, when cut into aud squeezed, yielded a considerable quantity of bright red frothy fluid; they were slightly congested and œdematous.

A quantity of dark venous blood escaped from the substance of the liver and kidneys when they were cut into.

The bladder was empty and contracted.

The intestinal vessels were engorged with dark fluid blood.

The blood-vessels of the retina with their branches were easily recognised with the naked eye, and contained fluid blood, as when touched the blood flowed along in them. The veins seemed full, but the arteries only half full.

PRUSSIC ACID.

At 3.3 P.M. Two minims of dilute hydrocyanic acid (B. P. solution) were injected under skin of back.

At 3.10. Respirations 84 per minute, pulse 240 per minute.

At 3.15. Two minims more were injected.

At 3.20. Respirations 72, pulse 300.

At 3.25. Respirations 76, pulse 320.

At 3.30. Ten minims more were injected.

It had two convulsions immediately after injection; its right ear was raised, it gave two cries, one after the other, and gasped in inspiration.

At 4.40. Respiration slow and almost imperceptible. Pupil was contracting slowly.

When its tail was pinched it gasped a little.

At 4.44. Pulsations of heart could not be detected. It was apparently dead.

The blood-vessels of the retina remained as they had been during the whole observation. The colour of the choroid remained the same, and there was no break in the blood columns of the retinal vessels or their branches.

Autopsy at 11.0 o'clock A.M. the following day:

Brain: The blood-vessels in the pia mater were numerous and large, and it had a very congested look.

Heart: The left ventricle was contracted and empty.

The right ventricle and right and left auricles contained black clotted blood.

The lungs were normal, and were not œdematous.

The kidneys were normal. The liver contained a few small tubercular deposits.

The eyes were normal. Only in two vessels of the retina could a small portion of them be seen empty.

Rigor mortis was not present.

STRYCHNINE.

A rabbit was injected, under the skin of the back, with one-seventh of a grain of strychnine; in two minutes after it had a convulsion and died. The retina and choroid were under view, but no change took place in the circulation, although the ears got warm and their blood-vessels full.

On the following day, the brain on examination looked normal. Its heart contained clotted blood in all the cavities.

The lungs, liver, and kidneys were normal, and the bladder was full.

HYOSCYAMINE.[1]

At 4.10 P.M. Half a minim of hyoscyamine was injected under the skin of a large rabbit.

At 4.16. Both pupils were widely dilated, and the blood-vessels of its ears were full.

It made a short struggle.

At 4.22. Respirations per minute 88.

At 4.25. No change from the beginning of observation took place in the retina or choroid.

At 4.37. Respirations 104, pulse 280. Makes short struggles now and again.

At 4.45. Respirations 97—sometimes hurried, pulse 240.

No change has taken place in the internal circulation of the eye.

At 4.55. One minim more was injected under skin.

No blood-vessels disappeared that were first observed, and no more came into view ; and the curves in the blood-vessels did not alter in the slightest, nor did any hæmorrhages take place.

[1] There was no atropine used in this case to dilate the pupil.

At 5.5. Respirations 102, pulse 240.

At 5.10. Two minims more were injected.

At 5.23. Respirations 80, pulse 270.

At 5.30. Three minims more were injected.

At 8.30. Three minims more were injected.

At 9.0. Respirations 48.

There was no change in the hlood-vessels.

At 9.20. Respirations 60, pulsations cannot be felt.

At 10.10. Died.

Before death the pulse was 140, and again 120 per minute. It was very intermittent. The respirations fell to ahout 40 per minute.

No change took place in the blood-vessels of eye after death, they remained of the same size as during life, and there was no break in the columns of blood in the retinal vessels.

The choroid retained the same hue as when the animal was alive.

Autopsy at 11.0 o'clock A.M. the following day :

The memhranes of the hrain were found congested, but the hrain suhstance when cut into was normal.

Heart : The left ventricle and auricle contained a quantity of dark fluid, blood, and clots.

The right ventricle and auricle contained dark fluid hlood.

The lungs were œdematous.

The kidneys were congested.

The liver appeared normal.

The hladder was full.

On dissection of the eyes the blood-vessels were seen to he normal, and just in the same condition as when examined with the ophthalmoscope.

MORPHIA.[1]

At 8.20 P.M. Respirations 120 per minute, and pulse 152.

Three grains of morphia were injected under the skin.

The rahhit's ears kept cool, and their hlood-vessels were not full.

At 8.35. Respirations 104, pulse 160.

At 8.40. Three grains more were injected.

Its ears still kept cool and pale.

At 8.45. Respirations 80, pulse 168.

At 8.50. Three grains more were injected.

No change whatever took place in the hlood-vessels of the retina or choroid. Its ears were cold and pale.

At 9.0. Respirations 96, pulse 172.

Three primary, two secondary, and twenty-two smaller hranches of retinal vessels were kept directly under ohservation the whole time.

At 9.15. Respirations 136, pulse 208.

At 9.20. Three grains more were injected.

At 9.35. Respirations 152, pulse 220.

The muscles of chest were convulsed occasionally.

[1] $\frac{1}{30}$th minim of hyoscyamine was used to dilate the pupil instead of atropine.

At 9.45. Respirations 140 and shallow, pulse 256.

At 9.55. Three grains more were injected.

At 9.58. Respirations 160, pulse 264.

At 10.0. Respirations 180.

At 10.10. Three grains more were injected.

At 10.18. Respirations 96.

At 10.25. No change in the circulation of the eyes.

The ears kept cool and pale. The pupils kept widely dilated.

At 10.28. Died.

The pupils remained dilated after death.

The choroid retained the same glow it had during life, and the vessels of the retina remained of the same calibre, but some time after death the blood in the largest branches showed a tendency in some parts to separate from the walls of the vessels.

Autopsy at noon of the following day:

Brain: The sinuses of dura mater were distended with dark clotted blood. The blood-vessels of the pia mater were numerous and congested.

Eyes: The pupils still remained very much dilated. The blood-vessels of retina did not look at all contracted, but the blood columns in most of them had broken and separated into detached portions.

Heart: The left ventricle was contracted and contained a very small dark clot.

The right ventricle was distended with a very large clot, the greater part of which was firm and decolorised.

The right and left auricles contained dark clotted blood.

The lungs were œdematous and congested, but small portions floated in water.

The liver and kidneys were normal.

The bladder was full.

Rigor mortis was present.

MORPHIA AND PICROTOXINE.

At 1.55 P.M. One-third of a grain of morphia was injected under skin of back of large powerful rabbit.

At 2.15. Respirations 36, pulse 220.

Its ears were pale and cold.

At 3.0. Respirations 60, pulse 180.

At 3.30. Respirations 24, pulse 152.

One-fifth of a grain more of morphia was injected.

At 3.45. Respirations 28, pulse 160.

One-fifth of a grain more was injected.

At 4.0. Respirations 24, pulse 168.

One-fifth of a grain more was injected.

At 4.15. Respirations 16, pulse 144.

One-fifth of a grain more was injected.

At 4.30. Respirations 10, pulse 108.

Three-fifths of a grain more were injected.

Ears remained cool and pale.

At 7.10. Respirations 16, pulse 150.

Two-fifths of a grain more were injected.

At 8.0. Respirations 20, pulse 120 and irregular.

At 8.15. Three-fifths of a grain more were injected.

At 10.0. One grain more was injected.

The blood-vessels of the retina and choroid did not alter in any way during the whole time.

About 11.0 P.M. the rabbit was released from the rest and got some bread and milk. It was sensible and could crawl about.

The following morning it seemed all right, but drowsy.

At 1.30 P.M. Respirations 40, pulse 320 and regular.

At 1.55. One-tenth of a grain of picrotoxine was injected under skin.

Its ears were cool, and the blood-vessels in a medium condition as regards quantity of blood in them.

At 2.30. Respirations 60, pulse 240 and regular.

At 2.45. One-fifth of a grain more was injected.

At 2.55. One-tenth of a grain more was injected.

At 3.0. It had two short struggles.

At 3.15. One-tenth of a grain more was injected.

Pulse 320. Breathing very quick and spasmodic.

At 3.30. It was very severely convulsed, and had twitching of the eyeball.

At 3.35. Its right ear became erect.

Had a severe convulsion.

At 3.40. Died in a convulsion.

No change whatever took place in the circulation of the retina or choroid, neither when the rabbit was at rest nor during the convulsions.

Two minutes after breathing ceased the pupil quickly contracted, and the columns of blood in both the veins and arteries suddenly broke up into detached portions, giving the same appearance as a column of mercury broken into numerous parts in the stem of a thermometer; this extended even to the small branches. No change took place in the colour or size of the blood-vessels after death, and the choroid appeared the same as when the animal was alive.

Autopsy at noon the following day:

The brain and its membranes were normal.

The lungs were normal.

Heart: The left ventricle was contracted, but contained a little dark clotted blood.

The right ventricle contained a firm dark clot, a portion of which was pale; the right auricle contained a firm dark clot, and the left auricle contained a dark clot.

The eyes were both in the same condition as seen with the ophthalmoscope.

NITRITE OF AMYL AND PICROTOXINE.

A small white rabbit (albino) was used.

Respirations 56, pulse 224. Its ears were cool and moderately pale.

At 9.5 P.M. Twenty minims of nitrite of amyl were placed on a piece of cotton, and the rabbit was allowed to inhale the vapour. In a minute

after the ears got hot and their blood-vessels intensely injected, but no change took place in the retinal or choroidal circulation.

Respirations 52, pulse 252.

At 9.8. Three-tenths of a grain of picrotoxine were injected under the skin of its back.

At 9.16. It was convulsed. Its ears were pale and cold.

Neither the choroidal nor retinal vessels were affected in any way; they remained the same as when first observed.

At 9.30. The convulsions were almost continuous. The ears kept cold and pale. The tail was spasmodically moved up and down.

At 9.40. Both during the convulsions and in the intervals between them the circulation was unaffected in the choroid and retina.

The ears kept cold and pale.

At 9.42. Died.

The pupil of right eye contracted after death, but the pupil of the left eye, which had no atropine instilled into it, remained somewhat dilated.

After death the choroidal vessels of right eye gradually emptied, till half an hour after death they only retained sufficient blood to show their position and allowed a pale reflection from the sclerotic. In the left eye the choroidal vessels retained more blood and the fundus did not appear so pale as in the right eye, but had more of a pink tint. Small vessels were seen in the disc of the left eye after death.

The columns of blood in the retinal vessels were not broken, nor the vessels smaller in calibre, and all the branches could be made out after death that had been observed when the animal was alive.

Half an hour after death post mortem rigidity was present.

Autopsy at 4.15 P.M. on the following day :

Rigor mortis was present.

Both pupils were equally and somewhat dilated.

There was a considerable amount of dark clotted blood in the sinuses of the dura mater. The brain substance was not congested.

The blood-vessels of the retina and their branches were full, and could be easily recognised. The choroidal blood-vessels were nearly empty ; near the centre of the fundus in both eyes they contained more blood and were well seen.

Heart: The left ventricle contained a little black clotted blood.

The right ventricle was distended with dark clotted blood, and the right and left auricles were full of dark blood.

NITRITE OF AMYL AND ATROPINE.

At 3.0 P.M. Respirations 124, pulse 288. The respirations five minutes after had fallen to 92.

Its ears were cool and their blood-vessels moderately full.

At 3.30. Twenty miuims of nitrite of amyl were placed near its nostrils on a piece of cotton, and in a few seconds its ears got intensely injected and hot, and the rabbit struggled, but no change took place in the vessels of the fundus of the eye.

Respirations 124, pulse 320. The ears got gradually cooler, and the blood-vessels smaller and less injected.

Some cotton was tied on the end of a stick with a few drops of nitrite of amyl on it, and the ears of the rabbit were placed so that they could be viewed at the same time as the eye, while looking through the hole in the mirror of ophthalmoscope. When the cotton was held close to the rabbit's nose, in a few seconds the ears flushed, got hot and injected, and remained so for a few seconds, when the nitrite of amyl was withdrawn; then got gradually cool and pale again, but no corresponding changes occurred in the circulation of the retina or choroid. When the cotton was applied again the ears took a minute and a quarter to get hot and injected, and in another minute after the amyl had been withdrawn they were cool and pale again.

The rabbit was left at 5.0 o'clock.

At 8.20 P.M. Respirations 114, pulse 280. The rabbit was all right again. It had micturated and defecated in the interval. Its ears were cool and not injected.

The vessels in eye were normal.

At 8.40. Respirations 98, pulse 272.

At 8.48. Six grains of sulphate of atropia were injected under the skin of its back.

At 8.55. Respirations 115 and shallow. Heart's action was too weak and fluttering to be counted. It was scarcely sensible to pricking, and when blown on or touched only occasionally moved.

At 9.8. Ears warm and extremely injected.

It was convulsed. It gasped spasmodically.

At 9.15. It struggled and died.

None of the blood-vessels were lost sight of, and no break took place in the columns of blood.

Autopsy, twenty minutes after death :

The brain was very pale, and there was little blood in the vessels of the membranes.

The ears were pale and bloodless.

The pupils were partly dilated.

The blood-vessels in eyes did not show any lessening in calibre nor break in continuity of blood.

The heart showed very slight contractile movements when touched or pricked. Each of its cavities contained a little dark-coloured blood.

Peristaltic action in the stomach and bowels continued, most markedly in stomach and least in the colon, and was only observed in the latter when it was touched.

The bladder was full of urine, and the bowels full of fæces.

ATROPINE AND PICROTOXINE.

At 3.0 P.M. Respirations 64, pulse 264.

At 3.30. Respirations 90, pulse 264.

A quarter of an hour after the light was reflected into the eye the pupil dilated a little and remained as if at rest.

Its ears were cool and pale.

At 3.45. One grain of sulphate of atropia was injected under the skin.

In two minutes after, the pupils were widely dilated, and the ears very pale and cold.

At 3.49. Respirations 92, pulse 300.

At 3.55. It struggled, and its ears got intensely injected and hot.

At 3.58. Respirations 60, pulse 256.

The inspirations and expirations were very shallow.

At 4.5. Respirations 56, pulse 256.

At 4.15. Respirations 56, pulse 260.

Its ears got paler. It struggled occasionally, and the respirations were still shallow.

At 4.17. One-third of a grain more of sulphate of atropia injected, and within a minute its ears got intensely engorged again.

At 4.26. Respirations 56, pulse 304.

Blood-vessels of ears not quite so full.

At 4.30. One grain of picrotoxine was injected under the skin. The blood-vessels of its ears immediately got intensely injected and the rabbit struggled.

At 4.39. Respirations 120, pulse 240.

At 4.42. It was convulsed. Its ears began to get pale.

At 4.43. Had a severe convulsion.

At 4.43½. The ears were nearly empty of blood, and cool.

At 4.45. Its right ear became erect. The convulsions became continuous. The ears kept quite cool and their blood-vessels not quite empty.

At 4.48. Its ears were cold and their blood-vessels almost empty.

The convulsions almost continuous, but no change whatever took place in the retina or choroid.

At 4.48½. Died in a convulsion.

The pupils contracted firmly.

Its ears remained the same after death.

At 5.5. The blood-vessels of retina were smaller than when the animal was alive.

The choroid, if anything, got a little paler in hue, but did not lose the brilliant red glow.

At 8.20. A small portion of the main trunk of one of the arteries contained only sufficient blood to make it visible; the rest of the trunk with its branches contained almost as much blood as when the animal was alive. The main trunk of the corresponding vein was almost empty in the greater part of its course, but all the branches of the vessels were seen. The small vessels in the disc were seen also.

The retinal veins and arteries of the left eye were very distinct; they had no breaks, and only in some portions of their course were they seen less full of blood than during life.

Rigor mortis was present.

Autopsy at 11.0 A.M. the following day:

There was a considerable quantity of fluid blood in the sinuses of the dura mater, especially at the base.

The brain was pale.

A considerable quantity of venous blood escaped from the orbits on removing the eyes. On dissection, the blood-vessels were just the same as when seen with the ophthalmoscope. The pupils were more dilated.

Heart: Left ventricle was partly contracted, and contained a little fluid blood.

Right ventricle was dilated with black soft clot, and the right and left auricles were full of dark blood.

The lungs and liver were normal.

The kidneys were slightly congested.

The bladder was full.

The blood-vessels of the intestines were very much engorged with venous-looking blood.

Rigor mortis was passing off.

HÆMORRHAGE.

The left eye of a rabbit was observed while it was put under the influence of chloroform by inhalation. It soon showed insensibility to touching its eyeballs and pricking its skin, although when its tail was pinched it gave the peculiar cry as in chloral poisoning.

The pupil contracted slightly from the influence of the chloroform, but got completely dilated after a few more drops of atropine solution were instilled into the eye.

Chloroform was administered till the rabbit ceased to cry when its tail was pinched.

Its ears kept moderately cool and pale.

Still no change took place in the retina or choroid.

The jugular and carotid vessels in the left side of its neck were snipped through, and the rabbit died in a few seconds. As the blood flowed from the wound the retinal arteries and veins quickly lessened in calibre, and some of the very smallest branches of the arteries and veins became empty and were lost to view; a number of the small branches, however, were seen. The main trunks of the vessels appeared reduced to about half the size they were when the animal was alive.

The choroid also became paler in colour, and its pigment appeared more distinctly.

The disc assumed quite a pale, cadaverous look.

Both pupils kept dilated—the left most so.

An hour after death there was no break in the continuity of the blood columns in the retinal vessels; those in the right eye were of the same size as in the left, and the choroid and disc were of the same hue in the one eye as in the other. The hæmorrhage from the divided vessels in the left side of the neck seemed to reduce the quantity of blood in the vessels of both eyes in exactly the same degree.

Rigor mortis had not set in an hour after death.

Autopsy three hours after death:

Rigor mortis was present.

Both pupils were alike in size and were in a medium state, neither contracted nor dilated.

The blood-vessels of the ears were not quite empty.

The sinuses of the dura mater were empty.

The vessels in the pia mater were very small and not nearly full.

The brain substance looked pale.

There was no blood in the orbits when the eyes were removed.

The liver looked normal.

The kidneys and lungs were pale.

Heart: The left ventricle was firmly contracted, and empty.

The right ventricle was contracted, and contained a small black clot.

The right and left auricles were empty.

The eyes, on dissection, had the blood-vessels in the same condition as when they were last seen with the ophthalmoscope.

The rabbits in every case were powerfully affected by the drugs, as seen by the respiration and pulse, but in no instance did the blood-vessels of the retina or choroid become altered in the slightest degree during the period in which the animal was alive. Even when the animals were seized with convulsions, when the vessels of the ears became intensely engorged with blood, or when they became remarkably empty, or when the one condition was produced immediately subsequent to the other in the same animal, there was not the slightest departure from the normal condition of the disc, retina, or choroid. No varicosity nor beading of the vessels took place, nor were they altered in the least degree in form. No pulsation was seen at any time in the veins or arteries of the retina; there was nothing like alternate filling and emptying of the vessels even in the case of death from rapid hæmorrhage.

Not till death occurred did any change take place, with the solitary exception of the hæmorrhagic case. In some even after death the vessels remained in the same state as during life, and it could not have been told from the appearance of the vessels that the circulation in them had ceased. In others the columns of blood in the retinal veins and arteries retained their size, or were a little smaller, but became attenuated in some portions of their course, especially at the edge of the disc where they bent over it, or in the part of a vessel where it crossed over another one: it was entirely a stasis or mechanical effect. No beading of the blood in the vessels occurred; in one case of picrotoxine poisoning the blood columns in the veins and arteries were seen to break into detached pieces, like 'bugle beads,' if the term may be used, as if the blood coagulated suddenly. This was in an old rabbit, and the vessels may have been less resilient, thus allowing the appearance described; the same has been seen after death in a human eye on dissection, but generally the other de-

scribed conditions existed. It only occurred in one of the cases of picrotoxine poisoning, showing that it was not a specific action of the picrotoxine.

The choroidal vessels in a few instances appeared unchanged; in most there was a gradual emptying—not always general; some vessels were completely emptied and lost to view, while others retained a little blood. Although the disc is considered to be mainly dependent on the cerebral vessels for its vascularity, and not on the ophthalmic artery like the retina, no change whatever took place in its appearance during life, and in a few cases there was no change after death, while in most it grew paler in proportion to the alteration in the hue of the choroid, as if the latter affected its appearance by refraction. In no case was it left so ghastly pale as in the rabbit that died from hæmorrhage.

Mr. C. Bader [1] states that Dr. Goodhart and he studied the effects of nitrite of amyl upon the blood-vessels in the healthy optic disc and retina. He says that in three or four seconds after taking three drops of the drug on sugar, the blood-vessels of the retina (arteries and veins, but especially the veins) become enormously dilated and gorged with blood, leaving no doubt as to simultaneously existing cerebral hyperæmia, with greatly accelerated circulation of blood.

To a man with good sight and eyes normal in every way, I gave five drops of nitrite of amyl on a piece of lump-sugar, after the eye had been observed for a quarter of an hour, and the pupil widely dilated with atropine; in ten seconds he said he felt it in his head, and in six minutes after owned to feeling all right again. No change whatever was observed in the circulation of the retina or the appearance of the disc or choroid. A quarter of an hour after six drops more of nitrite of amyl were given; in twenty seconds he felt it in his head, but the effect did not last so long, with the same negative result. Three days after, ten drops at once were given to him on a piece of sugar after the pupil had been dilated, and while the eye was kept under observation. His face and ears flushed, his conjunctivæ got slightly suffused, and his pulse rose to 100 per minute. He

[1] See 'Lancet' of 8th May, 1875.

said he felt it in his head, and it made his face feel very warm; but no change whatever took place in the circulation or the appearance of the disc, retina, or choroid.

I believe that cerebral congestion may exist without exerting any effect on the retina or choroid, and *vice versâ*. During the last five months I have been on the watch for optic neuritis amongst epileptics, but have not met with it, nor has the intra-ocular circulation appeared to me different from what I have observed in people in sound health, and never troubled with fits.

I have seen nothing in the eyes of any lunatics, not even in acute maniacs nor acute dements, that I could say was pathognomic, or connected in any way with the particular form of brain or mental malady from which the patient was suffering.

As regards general paralysis, so far from atrophy of the optic nerve existing in most cases, I have not met with it in one, nor have I yet seen a general paralytic blind before death from amaurosis setting in during his disease; they usually have remarkably acute vision. Good sight invariably remains to them long after their power of locomotion, and even articulation, is lost. There is at present in the West Riding Asylum an advanced general paralytic (T. G.), fifty-five years of age, who reads with ease and without spectacles No. 1 of Jaeger's test type. Another in the last stage (F. S.) can read No. 1 Jaeger easily, and even read unaided some photo-lithographic type one-third the size of Jaeger's smallest. Several make out No. 1 Jaeger; while others read No. 3 and No. 4 Jaeger, and are only prevented by the amount of presbyopia they have from reading the very smallest type without glasses.

Summary.—Not the slightest visible alteration took place in the disc, retina, or choroid, during the powerful action of the several drugs administered.

I have therefore come to believe that the appearances of the disc, retina, and choroid are unreliable guides to the condition of the brain; that the condition, as regards heat and flushing, of the face, ears, and conjunctivæ, and the state of the pulse, are infinitely more to be depended on as aids in diagnosis, while they are patent to, and can be observed by, every one.
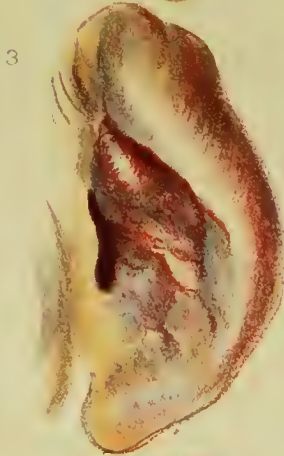
OTHÆMATOMA

OTHÆMATOMA, OR THE INSANE EAR.

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THE illustrated sheet accompanying this paper represents six typical cases of sanguineous tumour of the ear in its various stages, which were selected from thirty-two patients suffering from this disease, being all at one and the same time inmates of the West Riding Asylum. The drawings were taken from nature. On the day on which they were taken the building contained 707 male patients and 717 female patients; total, 1,424. The following table gives a list of the cases, with a statement indicating the relative frequency with which one or both ears were affected, and the particular form of mental disease with which each patient was afflicted :—

MALES.

Ward		Right ear	Left ear	Both ears	Disease
18	F. A.	—	—	1	General paralysis.
18	M. W. H.[1]	—	—	1	,, ,,
18	C. G.	1	—	—	Epileptic idiocy.
18	J. T.	—	1	—	Epiletic dementia.
18	O. O.	—	1	—	General paralysis.
2	J. S.	1	—	—	Chronic mania.
2	B. F.[1]	—	1	—	General paralysis.
2	J. W.	—	1	—	Chronic mania.
2	W. H. E.	—	1	—	Epileptic idiocy.
4	H. E.	—	1	—	General paralysis.
4	S. H.	—	1	—	,, ,,
4	J. B.[1]	—	—	1	Chronic mania.
14	J. C.	—	1	—	,, ,,

[1] These cases were those selected for pictorial illustration.

MALES—*continued.*

Ward		Right ear	Left ear	Both ears	Disease
13	R. P.	—	—	1	Chronic dementia.
13	J. L.	—	1	—	Chronic mania.
12	W. R.	—	1	—	General paralysis.
12	J. L.	—	1	—	„ „
9	J. W.	—	—	1	Epileptic dementia.
9	W. R.	—	1	—	Chronic mania.
9	J. H.	—	1	—	„ „
7	J. S.	—	—	1	„ „
7	G. L.	—	1	—	„ „
20	J. B.[1]	—	—	1	„ „
35	J. A.	—	1	—	Organic dementia.
		2	15	7	

FEMALES.

Ward		Right ear	Left ear	Both ears	Disease
26	C. N.	—	1	—	Chronic mania.
27	E. S.	—	—	1	Epileptic dementia.
29	A. C.	—	—	1	Chronic mania.
30	M. H.[1]	—	—	1	„ „
22	M. H.	—	1	—	Acute mania.
23	E. D.[1]	—	—	1	„ „
34	S. A. W.	—	1	—	Chronic mania.
32	C. T.	—	1	—	„ „
		—	4	4	

From the foregoing tables it will be seen that of thirty-two patients, the subjects of othæmatoma, twenty-four were males and eight were females. The disease existed therefore in 3·39 per cent. of the male patients, in 1·11 of the female patients; or in 2·24 per cent. of the whole number, irrespective of sex, who were at the time resident in the Asylum.

Of the twenty-four men, the right ear was affected in two, the left ear in fifteen, and both ears in seven instances; while of the eight women, the tumour appeared in the left ear in four, and in both ears in four cases.

From the statement kindly afforded me by Dr. Crichton

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[1] These cases were those selected for pictorial illustration.

Browne, as to the particular form of mental malady from which these patients suffered, it will be seen that no less than seventeen were the subjects of mania, acute or chronic, and five were epileptic. Eight patients were the victims of general paralysis (nearly all in an advanced stage), and only two suffered from dementia unassociated with epilepsy. It would appear then that othæmatoma is a disease which occurs for the most part in patients subject to attacks of a violent and paroxysmal character; that the left ear is very much more frequently attacked than the right (in the proportion of about three to one), while both ears are affected in about one-third of the cases. It may be further stated that when the tumours are present in both ears, the left ear is most frequently the first affected.

The appearance and course of these tumours can be well followed in the appended illustrations, concerning which it may not be uninteresting to remark that since they were made I have compared them with the admirable wood engravings of similar cases in Professor Gruber's ' Handbook of Ear Diseases,' and that there is a close correspondence in the appearances, not only of the early stages, but of the strange deformities afterwards resulting. The tumour is essentially one involving the vessels of the perichondrium of the external ear. It generally first appears in the helix, and is often limited to that portion of the ear, the boundary line of the anti-helix being well preserved. It may, however, commence at, or involve, the concha or the fossa triangularis, and Gruber mentions that in two cases the disease has been observed to commence in the meatur auditorius externus. The lobe or fatty portion of the ear is at no time involved.

Commencing as a tense, shiny, bright red or livid swelling, it may increase rapidly, and in about ten or twelve days develop to the size of a hen's egg. Its development is generally much more gradual, and the average dimension is about that of a hazel-nut or of a walnut. Sometimes it is quite even on the surface, as in the first figure, but much more frequently the ridges marking the fossa of the helix are preserved as seen in the second drawing. Most ob-

servers describe the pain experienced as much less than
would be the case in ordinary acute inflammation, nor is the
temperature very considerably elevated.[1]   One woman, men-
tioned by Gruber as the subject of othæmatoma, described
the sensation of heat 'as not greater than she had formerly
oftentimes perceived when the blood rushed suddenly to the
ears.' Of other subjective symptoms, neither deafness (un-
less the external meatus be very considerably occluded) nor
tinnitus are complained of.

The course of these blood tumours is very similar to that
of other hæmorrhagic extravasations; they may burst, and dis-
charging more or less disorganised blood, shrivel or refill, or
the contents may become coagulated and solidified without
bursting, or the tumour may spontaneously subside, though
not entirely, the ear never regaining its normal appearance
and shape. The peculiar characteristic of these cases is the
extravagant nature of the deformities which develop as a
result of the tumours, and is well shown in the two last
drawings appended to this paper. The lines by the side of
them indicate the length of the opposite (healthy) ears of the
same patients, and thus express the amount of shrinking
which may take place as a sequel of these cysts.

There is not much to be said as to the pathology of these
formations. Commencing as an extravasation, the effused
fluid is, if drawn off, found to be composed of blood or of its
constituents more or less disorganised. In one case mentioned
by Fischer, in his essay translated by Dr. Arlidge ('Asylum
Journal,' 1854, pp. 45–107), the tumour when punctured
discharged a fluid of a dirty cream colour, which later be-
came more bloody, much as is seen in the fluid drawn off
from cystic goitres. As a rule, however, the blood is not
much changed, and is seldom found to have lost its coa-
gulability.   The after deformity of the ear seems to be due
to development of fibrous tissue, to irregular adhesion of the

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[1] It should be mentioned that this account differs materially from that of
Dr. Alexander Robertson ('Glasgow Medical Journal,' July, 1875), who further
says that 'in exceptional cases the inflammation is so high that suppuration takes
place, and there may even be gangrene of considerable portions of the cartilage.
It is probable that suppuration or gangrene only takes place *after* bursting of the
cyst, and that it only occurs, as Gruber thinks, in purely traumatic cases.'

internal cyst-walls, and of the cyst itself to the adjacent car-tilage and skin. Not unfrequently new cartilage, secreted by the perichondrium, is irregularly added, and influences in a large measure, the variety of the supervening deformity. Examined after death the tumours are generally found to be composed of dense fibrous tissue, with shrivelled blood corpuscles, the fibres being stained at these points with blood tissue (Dr. Barlow in Dr. Alexander Robertson's paper). There is no justification for considering these cases as enchondromata, nor is there any evidence of atheroma of the coats of the vessels in patients the subjects of othæmatoma. Gruber suggests surgical treatment, and recommends evacuation of the contents of the tumours with after compression —a procedure not likely to be followed, I presume to think, in this country, when one considers the class of patients in which the disease occurs.

This brings one to the most interesting point in connection with these cases, their causation; and here I venture, as the result of inquiry from Dr. Crichton Browne and his able coadjutors, Drs. Lawson and Merson, as also on the authority of Dr. Langdon Down, to contradict some other writers on important points.

First, as to the idea of Dr. Wilks and those physicians who think with him, that these tumours are the result of external violence, either self-inflicted or by others. This surely must be at once dissipated for two reasons: one, that of all the many schoolboys whose ears are boxed, not one case has ever been recorded of a sanguineous tumour; and, further, that othæmatoma is no less frequent now when the treatment of insane patients is absolutely free from brutality, than it was in the days prior to Tuke and Connolly. If othæmatoma were caused by violence, we should see associated with it ecchymosis or actual hæmorrhage with rupture of the tympanic membrane, the last condition not uncommon, and of which I have seen several cases, as the result of accidental or intentional blows over the ear. Nor can we admit so unphilosophical a way of cutting the Gordian knot as the late Dr. Nicol suggested in his very interesting article on this subject ('Brit. and For. Med. Clin. Rep.,'

July, 1870), viz., that ' while refusing to acknowledge that extensive weakening of vitiation of the vegetative processes is invariably present, we must probably be content to allow the existence of some special weakness, such as is indicated by the term " blood dyscrasia." '

There is no doubt, however, that the state of acute excitement, often prolonged or of frequent recurrence, spoken of by this author in common with Fischer and Stiff, is an important factor.   Dr. Nicol also admits disturbance of the cervical sympathetic system.   This last is considered by Dr. Alexander Robertson as the sole cause of the disorder, and he quotes in corroboration of his view one most interesting case, in which a patient, aged twenty-two, suffering from acute mania of a few days' standing, was also the subject of exophthalmic goitre, apparently of much longer duration. On the morning after admission a peculiar red band made its appearance on the left side of the head and face, ' reaching from the junction of the hair with the forehead as far as the chin.   Both ears were red, and particularly the left one, but neither of them was very bright in colour.   There was obviously increased redness of the skin below the left ear, not, however, sharply defined like the band above described.'   It does not quite appear that this was a case of othæmatoma, and even if it were, the element of acute mania has to be added to that of the disorder of the vaso-motor system accompanying the goitre.   The interesting point is the occurrence of the band on the left side.

Although Dr. Nicol in his paper strongly enforces the rationality of such powerful elements of causation of othæmatoma, as the state of general excitement leading to functional disorder of the cervical and intra-cranial ganglia, and while further insisting on the presence of a special ' blood dyscrasia,' he considers that ' the mechanical factor is not to be overlooked.'   Speaking of the amelioration of the surroundings of the insane, he says (*Loc. cit.*, p. 195) :—' The ear is a part which benefits in quite a peculiar, if a limited way, by such amelioration.   During the hours of sleep, or at least of recumbency, while the head is supported on one of its sides, as it generally is for considerable periods with most

sleepers, and, moreover, frequently on one side more than another, the pinna of one ear, and more especially the anti-helix, the part generally affected, is pressed steadily between the mastoid part of the temporal bone and whatever happens to be beneath. A certain amount of derangement for the time of the processes of nutrition in the part must ensue, as well as reaction when the pressure is removed. With persons in ordinary health, neither of these processes is so energetic as to cause any evident structural change in the part; but with the presence of some dyscrasia of the sort already discussed, and with pressure equal to a large part of the weight of the head reacting from a stiff pillow upon the delicate and peculiar tissues of the pinna, we can hardly wonder that, in a patient suffering from prolonged mental disorder, serious structural lesions should result in the part. No doubt other and very various injuries and irritation of the external ear assist in individual cases, but this fact of pressure is present in every case.' The answers to this argument are very simple; namely, first, that there is no reason for considering the tissues of the pinna as delicate and peculiar, but that, on the contrary, its structure is that best adapted by elasticity and firmness not only to resist violence, but also to mould itself to receive pressure; secondly, that if pressure of the pinna against the mastoid process were the cause of hæmatoma, the tumour would appear on the surface which comes in contact with the skull, whereas the contrary is the case; thirdly, not to say anything of the presupposition of a uniformly ' stiff pillow,' there is not any case recorded in which there was abrasion of the skin, or other sign of friction or surface irritation. Nor is any evidence adduced to show why the left ear is so much more frequently affected than the right. As a matter of fact, however, while many people cannot sleep on the left side, quite as many persons lie on one side as the other, and rest to the full as well on the right as on the left side. Finally, Dr. Nicol seemed to anticipate such very obvious objections as the foregoing, for having decided that the factor of external irritation 'is sufficiently supplied in almost every case by the unfavourable position of the ear during the hours of recumbency,' he con-

tinues by saying that ' it need hardly be pointed out that the great variability within its own range of the physical factor, varying, as it does, with the shape of the ear, of the skull, the mode of wearing the hair, the usual decubitus, the accommodation of the patient, and so on—accounts for the inconstancy of this tumour, even in cases of excitement. In respect of natural and artificial covering to the external ear, women have generally a considerable advantage over men, and this may serve to explain the comparative infrequency of hæmatoma with the female sex.'

Another proposition requiring refutation, or at least further examination, is that these sanguineous tumours of the ear occur in mentally healthy persons. Gruber appears to consider that this opinion is now generally conceded, and in speaking of the subjective symptoms, casually mentions the fact that his knowledge is derived not only from insane persons in clear moments, but from ' three patients under his own care, who were absolutely healthy in mind.' (*Op. cit.*, p. 284.) While not presuming to say that so accurate an observer would have made a mistake in diagnosis of the local disease, it will be generally thought that the point is not so entirely set at rest as the Professor appears to believe. It would therefore be most desirable to have a succinct account of the disease or accident [1] from which these patients were suffering (for Gruber believes othæmatoma to be not a disease, but a symptom), as well as an accurate history of their mental condition. The result would not improbably be similar to that obtained by investigation of those brought forward by Fischer as cases of othæmatoma in sane persons. One was that of ' a very ignorant *wild* girl,' the other that of a man ' who had had *delirium tremens*, and was therefore transferred to an asylum for treatment.'

At this point it is interesting to note that, while Dr. Nicol asserts that ' hæmatoma is not recorded as in any way usual among idiots,' and Dr. Robertson ' does not remember

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[1] It is not denied by the writer that such tumours may arise as the result of injury, but in these cases the one would expect to find fracture of cartilage of the ear, with consequent hæmorrhagic extravasation. Such a condition was probably present in the case quoted by Dr. Wilks of a boy in whom a hæmatoma was developed as the result of a blow received while playing at football.

having seen it in idiocy,' two of the cases I saw in the West Riding Asylum occurred in epileptic idiots, and that Dr. Langdon Down, the greatest authority in this country on the subject, informs me 'that having kept accurate notes of the last 800 cases which have come under his notice, he finds that othæmatoma is met with in male congenital idiots in the proportion of 3·6 per cent. (*i.e.*, in even a larger proportion than in the 1,424 patients seen by me). Five cases occurred in the last 100 male patients. It is rarely met with in female idiots, and it is a point of some interest that it is seldom met with in accidental idiocy. Where the idiocy arises from developmental causes it may be found; where from causes operating in the early days of post-uterine life, almost never. The majority of these idiots who are the subjects of othæmatoma are epileptic; in all the element of excitement is excessive. Dr. Down further finds that the left ear is the one most frequently affected, in the ratio of 3 to 2.'

The following conclusions may be considered to have been fairly established as the result of much independent investigation :—

1. That prior to the occurrence of an othæmatoma the tissues of the auricle undergo a softening process (Virchow), or chondromatic degeneration (L. Meyer). These changes are synonymous with the 'vegetative disturbances' of Fischer and Nicol. No evidence has been adduced of the pre-existence of atheromatous disease of the vessels, as is believed by Gruber and others.

2. That the general nutritive derangement to which all othæmatomatous patients are subject, and the conspicuous absence of these tumours in the persons of patients suffering from monomania—a mental disease which involves only a portion of the cerebral machinery, or in which, as in melancholia, the lesion is of the most airily material nature (Nicol) —induce a belief that the aural tumour is in a large measure the result of a general, and not of a purely local, condition.

3. That intense general excitement is an important and almost universal factor in the causation of these tumours, leading as as it does to considerable vaso-motor disturbance,

and that the intimate connection of the cervical and intra-cranial sympathetic ganglia with the vessels of the auricle strongly predisposes to vascular extravasation in this neighbourhood. Dr. Robertson's case, already mentioned, bearing on this point is of great interest.

4. That the vascular distribution of the part, receiving as it does branches from the terminal arteries of the external carotid, all freely communicating with each other, and anastomosing with vessels supplying the brain structure itself, is sufficient to account for the preference of the auricle as the point for effusion. The helix being the thinnest portion of the external ear, is that part which is first attacked.

Lastly, that the left ear is most frequently affected, or where the hæmatoma is bilateral, is the first in which a tumour is developed. My own explanation of this fact is the nearer position of the left common carotid to the heart, and the more direct and less impeded arterial supply to the left than to the right side of the head. It may just be noted that arachnoid cysts, so frequent in that form of mental disease in which othæmatomata may be expected, are more commonly found on the left than on the right hemisphere of the brain.

P.S.—During the passage of this paper through the press the following case has come under my notice, and is worth recording as a type of patient who, though not insane, may be expected to be liable to sanguineous tumours of the ear. It is interesting to note the absence of pain, the sensation of heat (also described by Gruber), and the comparatively slight increase of redness of the skin.

John S., æt. 31, superintendent of a city mission house, consulted me at the Central London Throat and Ear Hospital on account of an increasing swelling of each ear.

He stated that, since the age of sixteen, he had devoted himself to mission work, that during the last five years he had held three or four services a day, and in the last twelvemonth had preached as often as five times daily. His general health was good, but he became much excited during his work, and had always been of a most excitable temperament, 'throwing his mind into all he did with all his might.'

Locally he complained of no pain or singing in the ears, nor was he deaf. He had noticed considerable heat and tingling in the ears for some time. This sensation was greatly increased by the slightest change of temperature, and after preaching. He had only remarked an alteration in the shape of his ears during the last twelvemonth. The swelling of each ear had increased rapidly in the last three months, and had latterly become noticeable to others. He had never suffered from any fits, nor was there any history of brain disease in the family. He had suffered no injury of the ears.

On examination there was found a swelling of the size of a large hazel nut of the helix of each ear protruding on its outward aspect as well as towards the skull. The general appearance was much as that of fig. 1 of the illustrations of this paper, except that the tumours were of the same colour as the rest of the ears, which were markedly red and flushed, the general complexion of the patient being very pale. The temperature of the ears was decidedly in excess of the rest of the body. No doubt could exist that the tumours were of the nature of othæmatoma.

MORBID HISTOLOGY OF THE BRAIN
IN THE LOWER ANIMALS.

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WHETHER, in the lower animals, changes take place in the nerve elements of the brain and how far such changes, if they exist, correspond with or differ from those observed in the human organ under morbid conditions, are questions which, so far as I am aware, have never hitherto been studied, and to which consequently no answer has been given. That this should be the case need not, indeed, excite surprise, when it is remembered that, even in health, the comparative histology of the brain in the lower animals has not received anything like that extended and searching investigation which its undoubted interest and importance deserves, and still offers a field almost untrodden by the step of scientific inquiry. I say this with every respect for the observations of such men as Beale,[1] L. Clarke,[2] Meynert,[3] and others, who amid their most extensive and invaluable labours in other departments of histology, have nevertheless not neglected this. The subject, however, is a very wide one, presents dangers and difficulties on every side, and both labourers and labour are needed. But as the difficulties are great, so will be the value of the ultimate results, provided these are the outcome of careful and accurate observation.

My thoughts and attention were first directed to the

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[1] Proceedings of Roy. Soc., June, 1863.

[2] Proceedings of Roy. Soc., September, 1863.

[3] Stricker's 'Human and Comp. Histology.'

subject of the possible histological changes which might occur in the brain of animals by my researches in connectiou with the subject of senile atrophy in man. It occurred to me that the laws of development and decline which I had there found to modify the nerve elements of the brain at different stages of life would probably hold good, to a certain extent at least, with respect to the lower animals; and this supposition was strengthened by a consideration of those changes and peculiarities which take place in the appearance, the habits, aud even the disposition of animals when advanced in years, and which are matters of common observation. It must be confessed, indeed, that the symptoms of senile dementia in the dog, for instance, present in some respects a remarkable similarity with those of the analogous condition in man, though manifested of course in an infinitely less variety of ways. Now in man the symptoms are plainly referable to a condition of atrophy of the cerebral hemispheres and degeneration of those nerve elements which form their most important constituent. I have, as regards the grey cortex, entered fully into the consideration of the histological condition present in these cases,[1] and while I should be the last to consider the subject as fully exhausted, I venture to think that my observatious, founded as they are on a large series of specimens, may be considered, so far as they go, accurate and trustworthy.

I will not enter into the details of the paper referred to ; it will suffice for my present purpose merely to recall some of the most prominent facts. With regard to the nerve corpuscles it was seen that, tracing the course of the retrogressive change, the starting-point in most instances cousisted in a slight granular deposit or formatiou in the iuterior of the cell, which, gradually increasing, produced successively a bulging or deformity, atrophy of the branches, degeneration of the nucleus, ending in destruction of the entire corpuscle. With respect to the vessels and the neuroglia, chauges were described in both these structures; in the former, oue of dilatation with deposits of yellow grauules ou the walls; in the latter, a condition of atrophy and degeneration. It is to the

[1] West Riding Asylum Reports, vol. iv.

above conditions in the human subject, and especially the appearances which they give rise to, that I desire to call attention at present, and these will be best understood by a reference to the various drawings I have given. (Fig. 3, also W. R. A. Reports, vol. iv.)

It is in the condition of old age, as was before observed, that we should expect to find pathological changes in the brain of animals, and it is to the consideration of the brain of a dog, dying at an advanced age, that my subsequent remarks will, for the most part, be devoted. But in order to study the condition of the nerve elements in the aged animal with any advantage, it is necessary that side by side with it should be viewed and studied the structural condition of the brain of the animal when in full health, and before age has brought impairment of physical and mental power. And this done, it will be my endeavour to go a step further, and compare the appearances presented by the various structures of the brain in the aged animal with those described in the human organ under similar conditions. In this study will, I trust, be found the solution of the first of the questions with which my remarks opened, and in some measure, if I mistake not, that of the second also.

The first brain the examination of which I desire to record, was that of a long-haired terrier bitch which died at the age of thirteen years. Up to the last the animal was alleged to be fairly intelligent, but manifested towards the close of her life a good deal of ill-temper, at times even towards her own master, and of which she was previously never guilty. It would seem probable, however, that in this we have evidence of loss of memory and diminished intelligence, for this irritability towards her owner seemed to be only manifested from her failure to recognise him. With respect to the animal's physical condition it is to be remarked that shortly before her death she began to take fits, which, from the descriptions given, seem to have been epileptic, and further, that she was partially paralysed in her limbs. I am unable to account for this paralysis (unless it was the result of the epileptic paroxysms), and with regard to the latter I would remark that occasionally in the human subject, as is

well known, epilepsy comes on in old age associated with senile atrophy of the brain. I am not aware, however, that the connection indicates any special pathological condition, that is to say, that any such has been discovered.

To external appearances the brain of the animal presented evidence of a slight amount of wasting, confined apparently to the anterior portions of the hemispheres. In these regions the membranes were somewhat opaque, and separated from the cerebral surface by serous fluid. In other respects, however, the brain to the unaided sight appeared free from disease.

Histological examination in this case put it beyond doubt that the grey cortex of the hemispheres was morbidly affected, and presented an appearance which, when contrasted with that observed in the middle-aged healthy animal, was at once most striking.

With regard to the individual elements, the nerve-cells first claim attention. Now a very short examination sufficed to show that in the case of the aged animal these bodies were, in the large majority of cases, surrounded by a clear space or gap in the brain tissue. The appearance was not universal, however, and was observed for the most part in relation with the large corpuscles of the deeper layers. It was at once evident that these spaces, surrounding to a greater or less extent the nerve corpuscles, were due to a condition of atrophy or shrinking of those bodies, and was not the result of any artificial change induced by the method of preparation. For, in the first place, they were not observed in any of the sections of the healthy specimens, prepared after an exactly similar method, and this alone I consider to be quite conclusive. But, further, the fact was soon brought out that, with regard to the bodies in which shrinking had taken place, and in some instances also in which this was not present, a morbid change in the character of the corpuscle was to be observed. The lesion consisted, with some apparent exceptions to which I shall afterwards refer, in a deposit of yellow granules in the interior of the corpuscle. The extent of this deposit or degeneration was very varying in degree, and gave rise to considerable dif-

ference in the appearances presented, according to the stage
of the degenerative process. In some there was merely an
accumulation of yellow granules, not sufficiently extensive to
occasion any great alteration in form, the nucleus and some-
times the branches of the cell being still intact. (Fig. 1.)
But the change does not stop here. Not infrequently the
corpuscles presented one uniform mass of yellow granules,
the nucleus having apparently quite disappeared. Between
this latter condition and the one before described, which
together represented, so far as I could make out in this case,
the earliest and latest stages of the destructive process,
numerous intermediate stages were observed, but all clearly
traceable to the same form of atrophy. One very important
change also I wish here to refer to, for it was very constant
and appeared to come on early, and this was a loss of some of
the poles or branches of the corpuscle. That this should take
place in the later stages is only what one would expect, and
could indeed hardly be otherwise, but that it should occur
in the early periods is, if correct, an important fact. There
can be no doubt that, with loss of its branches and connec-
tions, the function of the nerve corpuscle must be lost, but
then the question at issue is whether such atrophy of the
branches is a result of the atrophic process in the cell-body
tending to impair or abolish its function, or is in itself the
commencement of the mischief. I am not at present satisfied
as to the actual state of the case, but I may remark that not
infrequently I have seen several branches going off from a
nerve corpuscle which appeared to be entirely converted into
yellow granules, so that it is evident, at any rate, that great
changes *may* take place in the cell without involving destruc-
tion of the branches.

In those occasional instances in which a form of cell
degeneration other than that above described occurred, and
to which I formerly alluded, the change seemed to be one of
pigmentary degeneration, the cell being transformed into a
mass of dark-coloured granules lying almost loose in the
brain tissue, and having the appearance so well described by
Dr. Lockhart Clarke relative to the condition present in a

case of general paralysis, as 'a heap of granules ready to fall asunder.' [1]

With regard to the blood-vessels, I could detect nothing abnormal either in their course or in their structure, but the neuroglia was very decidedly altered. In health this substance may be said to be composed of two elements, one consisting of a delicate network of very minute fibres, the other being the granular matrix which is everywhere apparent between the nerve elements. Now the latter, in health, is extremely delicate and almost ·transparent, so much so that its existence in the form in which it is usually described has by some been denied altogether, and by others attributed to post-mortem changes. There can, however, be no doubt that under many morbid conditions in the human subject, and very decidedly so in the case of the animal at present under consideration, it was abnormally coarse and opaque, the fibrillar network also being evidently in places broken up and destroyed.

I pass now to consider the relation which the condition as above described bears, first, with respect to that found in the young, but fully developed, animal; and, secondly, to that already alluded to as being present in the senile atrophy of the human brain. On the first of these questions I need dwell but very briefly, for many points in it have already been incidentally referred to, and moreover, because an examination and comparison of the accompanying drawings (Figs. 1 and 2) will, I think, convey more readily and accurately than any description those differences to which I wish to draw attention. It will be observed that, with regard to the nerve corpuscles in the younger animal (Fig. 2), they present no evidence of granular or pigmentary degeneration; they are not shrunken, but closely surrounded by the brain substance in which they lie, and their branches are numerous and distinct. The intervening neuroglia is very delicate, and the fibrillar network is plainly seen. In all these respects therefore we have presented a very decided contrast with those appearances described in the brain of the aged animal,

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[1] 'Lancet,' September, 1866.

and we are led directly to one important conclusion—viz., that in the dog, in old age, decided pathological changes occur in the nerve elements of the brain.

With regard now to the relation which the changes above described bear to those found in the human organ, it cannot escape observation that the resemblance between them is very great. In both we find a progressive granular degeneration and atrophy of the cells, which up to a certain point seem of precisely similar nature, and the same destruction of poles and branches, while in the neuroglia we have evidence of a similar morbid process. The question then arises, is the resemblance complete? To this I must answer, not in so far as the present instance is concerned. For while in many of the nerve-cells, in the dog, the degenerative process was much further advanced than that represented in the drawing (Fig. 1), I was nevertheless unable to detect that stage which I have described as the *last* in the human subject, a stage in which the heap of granules representing the nerve corpuscle itself breaks down, leaving nothing but *débris*. In the next place, the number of affected cells in the case of the dog was not so great as in man, while, however, agreeing with the latter in being most common among the largest nerve corpuscles. It will be observed that a very interesting question here arises, and that is, whether in the lower animals degeneration of the nerve-cells of the cortex ever takes place to the same extent as in man. It may be that in the case of the dog under consideration the morbid process was only arrested by the death of the animal, and that, had its life been prolonged, a stage of degeneration still further advanced would have been present. On the other hand, however, it may be that, in the lower animals, the nerve elements being less highly developed than in man, less change is *possible* in them compatible with life. Or, again, the mental processes in man being infinitely higher and more complex than in the highest of the brutes, the manifestation of such processes is attended at last with a proportionate effect on the nervous tissue. It is not in my power at present to answer these questions, the solution of which, if I mistake not, can only be found in the continued study of the effect of

age on the brain of the superior animals, and by determining whether any relation can be made out between the amount of such effects and the degree of intelligence of the animal. Doubtless the subject is a very difficult one, and beset with many sources of fallacy. It is very difficult to pronounce, with anything like certainty, on the relative degree of intelligence possessed by various animals, so mixed up as such intelligence often appears to be with that which is nothing but mere instinct or antomatic action. And, again, in order that comparison with regard to the effect of age may be accurate and just, not similar but *relatively similar* ages must be taken; for what is old age in one animal may be but middle age, or even youth, in another. As regards the question of relative intelligence, however, there should I think be little doubt in some cases, as for instance, as to the superiority of the dog over the sheep or rabbit; and if in a series of cases it could be established that in the dog, in old age, greater changes occur in the nerve elements of the brain than in the sheep or rabbit, I think it would go far to show the correctness of the theory before advanced—viz., that when the nerve elements are less highly developed and the functions they subserve less complex and extensive, the effects of age and prolonged use are not so apparent as in a more highly organised and active organ. I am in hope, however, that before long further investigation in the direction I have indicated may elicit the truth.

With regard to the vessels of the brain of the aged dog, I stated that, so far as I could make out, they were free from any morbid process or deposit; and in another case I shall have occasion to bring forward a similar fact—viz., a morbid condition of some of the nerve elements *without appreciable vascular disease.* I do not know how far this observation will be borne out by subsequent experience, but the fact as existing in these two cases is, I think, of considerable importance. So far as my experience goes, the vessels of the human brain, in cases of senile atrophy, invariably present to a greater or less extent some evidence of a morbid process, dilatations, deposits on the walls, &c.; but in animals, as I have shown, this may not be the case. In the second place,

the fact throws light on a question which has often been discussed with respect to morbid processes in the human brain, whether the vessels or the nerve-cells form the starting-point of such conditions, both structures being affected. I consider that the facts I have brought forward show conclusively that in animals there may exist disease of the nerve elements *without* any affection of the vessels, and if this be true with respect to animals, why not also in the case of man?

The next brain which I examined, with the object of ascertaining whether any morbid condition could be detected, was that of a horse which, according to the account furnished to me, had been killed at a very advanced age. While, however, it is my intention to enter on a brief description of some points of interest which it presented, I must add that I do not rely on the case as I do on that of the dog already given. The animal was said to have been, as before stated, advanced in years, and this indeed seemed probable from the general condition of the brain, as also, so far as I could judge, from the state of the teeth, &c. The exact age of the animal, however, I could not ascertain, neither could I learn whether any mental symptoms had been present, so that I cannot rely on the case being one of senile atrophy. With regard to the external appearances presented by the brain in this case, the membranes over both hemispheres were considerably thickened, and the pia mater strongly adherent to the subjacent cortical substance. There was slight wasting of the convolutions in the frontal regions. On cutting into the brain substance the cortex was observed to be unusually pale, but in other respects nothing abnormal could be detected by the unaided eye. Examination of fine sections under the microscope, however, showed that other morbid conditions really existed. The first and second cortical layers immediately underlying the adherent pia mater were decidedly affected, the structure of the first being loose and in some places broken down, while in many instances the small cells of the second layer, those which I have almost always found the least affected by general atrophic changes, were decidedly diseased. The drawing I have given (Fig. 5) re-

presents some of these cells shrunk and atrophied. In the deeper layers again the large nerve-cells presented the condition described as granular degeneration, but not so commonly or to such an extent as in the case of the dog before described. The vessels appeared to be healthy and free from morbid deposit of any kind, but the neuroglia again, in some places, was coarse and its texture degenerated. In this case therefore we have presented another instance of a morbid condition of some of the nerve elements, without perceptible vascular disease.

In the brain of a cat eight years old, which died suddenly, I could detect no morbid condition of either cells, vessels, or neuroglia, but an extraordinary proliferation of the connective tissue corpuscles in the pia mater. The intelligence and activity of this animal, it should be stated, had certainly been somewhat weakened, but not to any great extent, and there was no paralysis. I have not been able to observe this condition of the corpuscles of the pia mater in any other specimen of the cat's brain, and regard it as decidedly morbid.

In the course of the foregoing remarks, those conclusions which seem to spring from the present series of observations have for the most part been indicated, and there is no necessity that they should here be repeated; but while, so far as they go, they may it is hoped be found accurate, it must be added that the inquiry is at present merely in its first stage. The subject is, as far as I am aware, a new one, and from its nature can be investigated but slowly and under considerable difficulties. More and more, however, has it become apparent to me that it is one highly important as well as interesting, and well worthy of further study. It is indeed earnestly to be wished that advantage might be taken more generally than is done of those opportunities which from time to time occur of examining the brains of animals dying at a great age, or in which acute cerebral symptoms have been present, and thus advance our knowledge in this direction. Facts thus accumulated would, I feel sure, help towards the elucidation of some of those great questions which arise in connection with the functions of the brain,

and enable us to determine, with greater accuracy than we can at present, the significance of many pathological changes in the human organ. Unfortunately the labour of such an investigation is great, involving, as it does, an accurate knowledge of the cerebral structure in man and animals, as well as the means whereby such structure is to be demonstrated.

EXPLANATION OF PLATE.

Fig. 1. Section from the Frontal lobe of an aged Dog, showing degeneration of the nerve cells.　× 400.

Fig. 2. Section from the same regions in a young and healthy Dog.　× 400.

Fig. 3. Section from the Frontal lohe of the human hrain in a case of Senile Atrophy, showing degeneration of cells, &c.　× 400.

Fig. 4. Section of the healthy human hrain, showing multipolar nerve cells (Frontal lohe).　× 400.

Fig. 5. Section of the brain of aged Horse, Frontal lohe, showing atrophy of the cells and neuroglia.　× 400.

Fig 1.
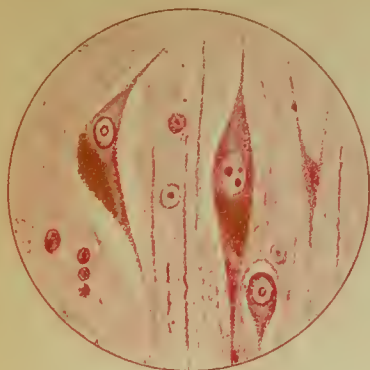
Fig. 2.
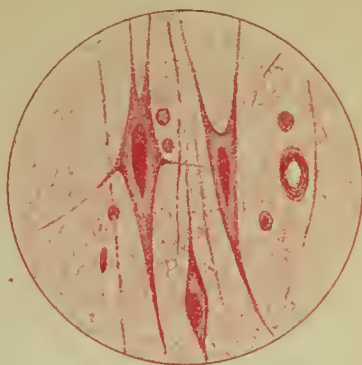
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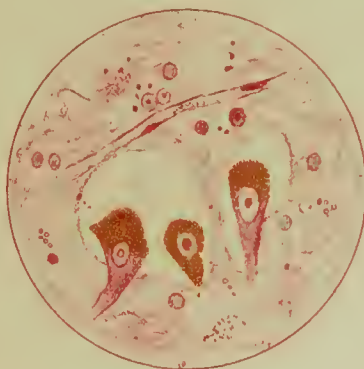
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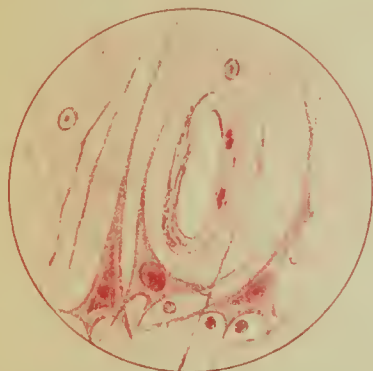
Fig 5
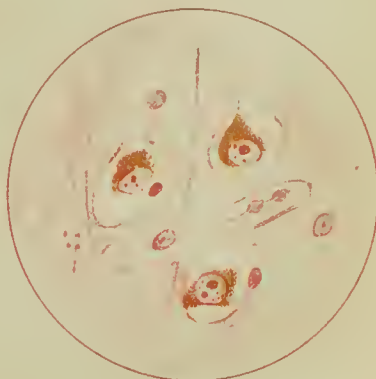
CEREBRAL HYPERÆMIA.

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In the present consideration of cerebral hyperæmia, the writer will follow the definition laid down by Handfield Jones, namely, that it is an increase in the amount of arterial blood passing through the encephalon. This is the only true hyperæmia, fulness of the veins being more properly described by the term 'venous congestion,' which is rather associated with arterial anæmia. Nevertheless, it is perfectly obvious that if there be an increased flow of arterial blood through an organ, there must also be an enlarged venous current; when, however, the veins become congested and the current is impeded from any cause, then the arterial flow is more or less obstructed. After this brief statement, the subject of venous fulness will be dismissed from the present inquiry.

Hyperæmia of the contents of the encephalon is a condition which may be truly physiological, or which may be decidedly pathological, and even destructive to life. As the first, we find it whenever the brain is bent to study or to thought; the effect of the will in so bending the attention is, so far as can be ascertained, to dilate the encephalic vessels, and increase the amount of arterial blood passing to the cerebral cells. The consequence of this is to endow the intellectual powers of the individual with an increment of energy or capacity, and, in doing so, to enable him to cope more successfully with the matter before him. The activity of an organ is in strict relation to its blood supply, and the capacity of each brain from time to time, not as compared

with other brains, depends upon the amount of arterial blood
passing into it. Such is the true physiological cerebral
hyperæmia of brain activity, contrasting with the anæmia,
which is an essential factor of sleep. As sleep comes on the
brain falls, becomes paler, and many of its blood-vessels
that could be recognised during the waking state become
indistinguishable. When consciousness returns in the act of
awaking the process is reversed, the brain fills, grows
ruddier, and the vessels, which were lost sight of in sleep, can
again be distinguished by their enlarged calibre. Such is
the difference betwixt the conditions of waking and sleeping
—so far as the blood supply goes—but no further. The
subtler changes in the cerebral cells themselves have not yet
come within the sphere of our cognizance. This much,
however, we already know, and that is, that in functional
activity the brain is highly vascular; in sleep it is anæmic,
and that abolition of brain function may be brought about
by arrest of the supply of arterial blood, either complete or
partial according as the vascular current is affected, and in
strict proportion to the diminution of the supply of arterial
blood. This subject has been dealt with at length in the
fourth volume of these reports (1874), in my article on
' Cerebral Anæmia,' and need not be further discussed.
Another division of this subject, namely, the possibility of
changes in the amount of arterial blood within the ence-
phalon by means of the peri-vascular spaces, is also con-
sidered there in detail, and such changes in the vascular
supply of the brain will be here assumed as demonstrated
and accepted. The reader, therefore, who thinks that many
matters are here taken for granted which may be held to be
doubtful, must refer to that article, as space will not permit
of their reconsideration here; even if it were expedient to do
so, which does not appear to be the case.

Having thus seen that there are normal and physiological
changes in the vascularity of the intra-cranial contents, we
can proceed to the consideration of those abnormal conditions
which are known as cerebral hyperæmia in its various forms.
Such change in the vascular supply is brought about in
several ways, and the conditions of each change are so dis-

similar, both in their pathology and in their indications for treatment, that it becomes desirable to discriminate betwixt the different forms in a clear and unmistakeable manner. This is the first step. On the very threshold of the inquiry we are brought face to face with the fact that there are two factors in the production of localised hyperæmia, namely, (1) an increase in the blood pressure generally—the vascular factor; and (2), a change in the tissues themselves, by virtue of which they attract more blood—this is the tissue factor. This latter occurs under two totally different sets of circumstances; it may be associated with functional activity, like the gastric vascularity of digestion, or it may be the means of repair, as seen in the vascularity which results from an injury. All repair involves an increased supply of nutritive material, which is brought about by arterial fulness.

It is too much the habit to assume that all modifications of function are merely questions of blood supply. We have become so familiar with the fact that activity of organs is accompanied by vascular fulness, and quiescence by comparative anæmia, that we are apt to lose sight of the fact that these vascular changes are but consequential to something which stands to them in a causal relationship. That localised hyperæmia rests upon a casual something which we have not yet unravelled, but which, we have good grounds for holding, exists in the tissues themselves, by whose agency they attract a larger flow of arterial blood, we have every reason to believe. It will not do to assume that all the difference lies in the vaso-motor and vaso-inhibitory nerves, and that by fulness or contraction of the local blood-vessels all can be accounted for. Even if this were all, what sets in motion those vaso-motor changes? There is much not yet within our ken; what we can do is to consider the subject so far as it has come within the limits of our cognizance.

It will, therefore, be well to divide the subject of cerebral hyperæmia into three sections. The first is the form found along with vascular excitement, the hyperæmia of vascular origin; the second is that form where there is encephalic fulness without vascular excitement; and the third is that form produced by the action of drugs. Of course

in practice these different forms overlap each other, the third form being most commonly seen in the effects of alcohol.

The first form of cerebral anæmia, which may fairly be denominated the vascular form, is found chiefly along with permanent changes in the vascular system, of which a high blood pressure is one of the most pronounced. This is the form of cerebral hyperæmia most commonly seen in the plethoric and middle-aged. Its most typical form is found with the following surroundings :—A florid red-faced person of stalwart physique, upon which, however, time is stamping his mark, complains of intense headache with sense of distension, and not rarely of a feeling as if a bolt were driven into the forehead. The eye is injected, and is highly sensitive to light, bright flashes of light being commonly seen, but which are entirely illusive ; there is also much intolerance of light. Hearing is affected, and noises are heard either entirely of centric origin, or exaggerations of real noises, with more than natural acuteness of hearing, or partial deafness. There are disturbances of senation of a more general character, and motor disturbances, convulsive movements, or incomplete paralysis. Thus speech is either very ready, or is muffled and indistinct. There are in fact those disturbances which are known as exaltation of function with more or less imperfect co-ordination, and incapacity to discriminate exactly. At the same time the pulse will be felt full, tense, and incompressible ; the heart's sounds good, the aortic second sound accentuated distinctly, indicating high arterial tension ; good impulse : indeed the evidences of ventricular hypertrophy and of a high blood pressure. The arteries will usually be found to be more or less atheromatous, and to give to the sphygmograph the characteristic square-headed tracing, long ere there is any change perceptible to the finger. Often there is a foul tongue and sluggish bowels.

Such is the true vascular hyperæmia of the plethoric. Its pathology is that of the changes so commonly found along with the gouty kidney. Long ago the connection of cerebral congestion with ischuria renalis was recognised, and Dr. Adair Crawford, in the ' Cyclopædia of Practical Medicine,'

says of ischuria renalis, 'The persons most subject to this affection are fat corpulent men, between fifty and sixty years of age, of a gouty habit, and naturally predisposed to cerebral congestion.' Rokitanski first pointed out the connection betwixt cerebral apoplexy, with clot, and hypertrophy of the left ventricle with diseased vessels.[1] Now we know that hypertrophy of the left ventricle without valvular disease is most frequently found amidst the sufferers from chronic Bright's disease, of the cirrhotic or gouty form. As it is impossible simply to give any fair conception of vascular cerebral hyperæmia without scanning briefly the complex condition with which it is usually associated, a brief review is necessary. Whether the starting-point be excessive indulgence in food, especially of a nitrogenised character, so that the kidneys are persistently called into high functional activity with its hyperæmia—a condition which is the common cause of a growth of interstitial connective tissue in the organ—or some cold has set up organic change in the kidneys, or other excitant of structural change is in action, the kidneys become less competent to discharge their duties; there is, in fact, a certain amount of renal inadequacy—that is the first step. Then follows arteriole spasm, and with it a rise of blood pressure. A rise in blood pressure evokes hypertrophy of the left ventricle in well-nourished persons, and thus the blood is driven into the arteries with greater force. Consequently the blood pressure is maintained abnormally high, and betwixt the hypertrophied left ventricle and the hypertrophied peripheral muscular ends of the arterial system, the arteries are highly distended, and this over-distension leads in time to tissue growth in the arteries, known as atheroma. Here is a distinct and intelligible sequence of morbid changes. The thin-walled vessels of the brain are apt to become much altered in time by this repeated high distension, and apoplectic rupture is most commonly found so related. But with that we have not here to do. The question here is the earlier stage of cerebral arterial hyperæmia, which not rarely is the procursor of such rupture.

[1] Vol. iii. pp. 398–9. The description is perfect; it was only left for clinical observers to explain the causation.

There are certain factors which affect and influence this local expression of a general condition. If the flow of blood to the head has been specially determined by much intellectual effort, by great demands upon the brain, or by indulgence in alcohol, then cerebral hyperæmia will be pronounced in the persons just described, and any acute disturbances in the vascular system generally will be felt most distinctly in the encephalic contents; while in others, an attack of dyspepsia, an eruption on the skin, an attack of lumbago, or other form of malady, may be the most marked evidence of the general disturbance. Lithiasis, or lithæmia, as this condition is denominated, consists of an overlading of the blood with azotised detritus, with nitrogenised waste, rather the product of peptones broken up in the liver into glycogen and nitrogenised refuse, than the outcomes of tissue metamorphosis, or histolysis. Consequently we find that the chronic condition has superimposed upon it at these times a state of acute exacerbation; that is, that the permanent state has periods of acute accentuation, and becomes more pronounced at these times. Consequently, too, the powerful ventricle acts vigorously against a blood pressure now higher than its usual height, which is much above the normal; the general fulness of the arteries is increased, and the pulse is full, tense, hard, and incompressible. The carotids are distended, the cephalic vessels are dilated, there are the evidences, physical and psychical, of cerebral hyperæmia, and vascular fulness of the contents of the encephalon. The condition is perfectly intelligible, and the dangers are obvious; they are either apoplectic rupture or serous apoplexy. The production of the first lesion under the above circumstances is too patent to need any comment or explanation. The second is not so simple. We are too much accustomed to associate serous apoplexy with venous fulness and arterial anæmia, especially when the latter is due to uræmia (Traube), to realise how commonly it is found along with cerebral hyperæmia. We know that when the cerebral arteries contract within their perivascular spaces, the vacancy is filled by lymph, and the association of serous apoplexy (Gehirn-œdem) with arterial anæmia is comprehensible enough. But the

production of serous apoplexy in cerebral hyperæmia has been hitherto incomprehensible, and has been wonderfully put out of sight, as inexplicable facts often are. I believe, however, that the explanation has recently been given by Dr. Batty Tuke in his Morisonian Lectures (1874). He says:—'It will be remembered that the arteries and veins of the cerebral hemispheres are enclosed in comparatively rigid cylinders of nervous tissue and neuroglia; that between the brain substance and the proper coat of the vessel we have a fine hyaline membrane surrounding and encapsulating it; and that by the space, small as it is, between the vessel and hyaline membrane, are conveyed the lymph products to the lymphatics of the pia mater. We have thus a main and an overflow, as it were, in one cylinder; the main, however, is elastic, and is liable to changes in its calibre to such an extent, that under their influence it fills completely the retaining cylinder. *When this takes place, the overflow is occluded and rendered useless.* That the artery does in hyperæmia fully occupy the cylinder, is proved by the fact that, in subjects in which congestion has been a frequent condition—*e.g.*, epilepsy and general paresis—the cerebral substance is distended, and the comparatively rigid cylinder enlarged from pressure from within. But this is to be seen only in old-standing cases; in recent ones the vessels appear to be, if you will excuse the paradoxical expression, fuller than they can hold. Under such circumstances, what becomes of those effete or superfluous matters and exudates produced by hyperæmia and stasis, which should have been carried off from the surrounding tissues, and which may be presupposed to be increased in quantity to the same extent as the blood supplied? They cannot find exit by their normal passages; accordingly, the brain substance and the membranes become œdematous; the pia mater becomes displaced by the serum, which, oozing through the tissues, raises it from the convolutions within and without the sac of the arachnoid; fluid becomes arrested, and the ventricles of the organ become dropsical.' After this graphic picture, the occurrence of serous apoplexy from cerebral hyperæmia becomes intelligible enough. Considering the flow of arterial blood towards

the cerebral cells, and the effects of the impaired lymphatic flow, leaving waste products in excess as sources of irritation in them, we cannot feel surprised at the great excitement which is exhibited by persons under such circumstances in the early stages of the disease. The general exaltation is only what might be expected from the facts of the case, and that serous apoplexy with obliteration of function, often fatal, should follow, is no matter for incredulity.

Under these circumstances the treatment of cerebral hyperæmia ought to proceed as follows:— The first great indication is to lower the vascular system, and with it the encephalic engorgement. With such a view venesection was much resorted to of old, and was doubtless an efficient measure. At present other measures suggest themselves, such as depressing the circulation by nauseants, as tartar emetic; or, better still, by vegetable vascular depressants, as aconite, calabar bean, &c., with free purgation. By such measures the force of the heart and the blood pressure within the arteries would both be distinctly lowered; the cerebral vessels would be less distended; and the lymphatics, compressed against their rigid walls by the dilated arterioles, would be relieved and resume their function. Thus not only would the intra-cranial engorgement be relieved, but the overflow would be re-established. After the circulation has been quieted, the excitement remaining in the cerebral cells might be composed by a dose or two of chloral hydrate, whose double action upon the cerebral cells and the circulation renders it peculiarly suited for the relief of such a condition. The general quiescence so induced is shown by the altered demeanour of the patient, in the comparative calmness of the circulation, and the absence of excitement generally. If there be, as there often is, a gouty element present, then colchicum might be added to alkaline saline purgatives with advantage. Cerebral hyperæmia is the pathology of those tornado-like gusts of passion and intellectual or emotional excitement so commonly seen in gouty subjects, especially in robust men over middle age.

II. We can now proceed to the consideration of that form of cerebral hyperæmia which depends rather upon changes within the encephalon itself than upon any modification of the vascular system generally. Here we find the circulation

calm, and sometimes even depressed, while there are all the symptoms of cerebral excitement combined with fulness of the intra-cranial arterial vessels.

This, the second form of cerebral hyperæmia, is found in a totally different class of patients to that form described above. The first form is usually seen in private practice; the second form is best studied in the wards of our public asylums. By the courtesy of Dr. Crichton Browne, ample opportunities for observation have been furnished to the writer in the large institution under his supervision, and on such observations the following remarks are founded.

Previous, however, to going into a description of this second form, it may be desirable to review a matter alluded to at the commencement of this paper, namely, the two factors of cerebral hyperæmia—(1) the changes in the blood-vessels; and (2) some change in the cerebral cells themselves, by which they become universally active, and, as part of this activity, attract blood with unwonted energy We have seen that the symptoms of cerebral hyperæmia include alterations in the sensations, and markedly in the special senses of sight and hearing. Intolerance of light and sound, flashes of light and loud noises in the ears, unrecognisable by others around, are marked symptoms of this pathological condition. It is not necessary in the present inquiry to bring in any other disturbances of function; these are amply sufficient for our purpose here. It is obvious that in these disturbances we have modification of function, that the sensations are exalted, that ordinary objects observed by the eye and ordinary sounds noted by the ear are all accentuated and magnified; and not only that, but that vibrations of light and sound waves ordinarily unnoted become vivid and distinct. This must depend upon an excited condition of the receptive cells themselves. But the hypothesis that this exaltation of function rests simply upon an abnormally free supply of arterial blood to these cells, and that this augmentation of receptivity is due to the increased amount of blood merely and solely, is not a sufficient explanation. The opposite hypothesis, that the increased vascular supply is due to cell activity, by which more blood

is attracted at the same time that the excited condition of the cells renders ordinary stimuli intense or intolerable, and impressions not ordinarily recognised by the senses of sight and of hearing distinct and perceptible, can be equally well raised and maintained. We must clearly recognise these two factors, even if we cannot always appreciate the proportions of the different factors, or appraise their exact value in every case. In practice, in many cases, the two factors are blended, and a complex condition is the result. Still it will usually be possible by care to discriminate to which of the two classes any case may belong. Doubtless excessive vascularity produces cerebral excitement and augmented cell activity ;[1] while, on the other hand, exalted cell activity is not without its effects upon the circulation, and especially upon the intra-cranial section of it.

Having thus laid down these two factors, and admitted that the two forms of cerebral hyperæmia may exist in a combined and blended form, we can proceed to the consideration of the second form of cerebral hyperæmia, where there is dilatation of the intra-cranial vessels without excitement in the vascular system generally. This may occur either in a form which does not involve mental aberration, or it may present itself in the guise of mania.

This form is usually the result of over-excitement, of mental toil, of surprise, or of some of the numerous accidental causes of cerebral congestion and excitement. There is here general restlessness, excited action, often great loquacity, with incoherence, and a broken, confused, jumbling together of thoughts, as if the thoughts got into each other's way in their tumultuous haste. Sometimes the ideas seem to centre themselves around some prominent leading thought, the centre-piece of the rotatory chaos, while at other times there is general excitement, with great volubility on no subject in particular. There is sleeplessness, because the anæmia, which is an essential factor of sleep, is impossible. Such are the noisy, excited patients so common in asylum wards, who are such an annoyance to other patients. If the form be compatible with sanity, the patient usually complains of

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[1] See Brunton on 'Digitalis,' pp. 67 and 69, where flashes of light, as bright spots, formed a part of the phenomenon of digitalis intoxication.

heat and fulness of the head, throbbing, mental confusion, with consequent depression—not the melancholia of cerebral anæmia—the face may or may not be flushed, and the pulse may be calm. The sleeplessness continues, and, together with the restlessness and muscular activity, sooner or later leads to exhaustion. In the shape of delirium tremens temporary cerebral hyperæmia, in the form of vaso-motor paresis from alcoholic intoxication, is far from uncommon. Often there is considerable vascular excitement, in which case the patient is almost as incessantly active as the acute maniac, indulging all day long in excessive muscular action, until a sufficient amount of fatigue has been induced to permit of sleep. In one case known to the writer, the patient used to turn out at daybreak and hunt his illusional demons up and down a long lane, till sheer weariness at night compelled sleep; while another used to go into some lonely fields, and erecting a leaping-bar (called in Northern phrase a 'cat-gallows'), from morning till night made running leaps over it until sleep became possible. In these cases the will could direct the discharges of the excited hyperæmic convolutions into certain channels; in other cases, where reason is unseated, we get the apparently aimless and objectiveless muscular activity of the excited maniac. In these conditions of cerebral excitement there is not only hyper-activity in motor convolutions, but there is also exaltation of function in the other convolutions, as in the sensory ones, which gives illusions as well as distortions of facts; and also in the ideational convolutions, producing hallucinations and insane thoughts. A part of this cell activity is the attraction of blood in unwonted quantities, and by this means the exaltation maintains and perpetuates itself. The effect upon the intra-cranial circulation is to dilate the minute arteries and increase the amount of arterial blood within the encephalon. If there be also general vascular excitement we get a very active condition, such as is seen in the acute maniac; but if there be vascular depression, the symptoms are not so acute. But after all it is but a question of degree. The patient who is so violent that he must be confined in a padded-room, will be found with a certain amount of excitement in his circulation, while the less violent patient in the wards, when examined,

will be usually found to possess a calm, quiet, and not rarely even depressed circulation. It is important from a therapeutic point of view to recognise this blending of the two factors in varied proportions in each case, as well as being diagnostically interesting.

During several lengthy visits to the West Riding Asylum, numerous opportunities have been furnished of noting cases of excitement with increased encephalic vascularity, where the circulation generally was calm and quiet. This was opposed to the *à priori* view that local vascularity of an active character must necessarily be linked with general vascular excitement; but such nevertheless is the case, as repeated observation has shown. Such cases are too common to need any illustrative case here.

While such cases undoubtedly exist to a large extent in asylum practice, they are not unknown in ordinary practice, and are usually the result of prolonged overwork of the brain. Niemeyer (vol. ii. p. 164, ed. 1870) writes : ' In this form of cerebral hyperæmia, which develops chiefly as the result of excessive mental labour, there is usually frequent pulse and other symptoms of fever, but in these very cases a greater amount of blood in the face, &c., does not correspond to the greater amount in the brain; the patients are not high-coloured, but are often even pale. From the fever and sleeplessness they rapidly lose strength, emaciate, and if they do not fall into the right hands they are in great danger of dying from their disease. Finally, the excitement gives way to apathy, the insomnia to deep sleep, from which the patient cannot be aroused, and in which they die. Far more rarely there is permanent mental disease.' Such are marked cases where the cerebral hyperæmia is associated with some general vascular excitement. In a less pronounced form such cases are not rarely seen as the consequence of protracted mental overwork. In these cases there is vaso-motor paresis of the intra-cranial vessels from exhaustion, but the general circulation is quiet; or if there be an increase in the frequency of the pulse, it is counterbalanced by loss of force; there is a low blood pressure, with dilated vessels, especially those of the nerve-centres. In these conditions, depressant measures

are distinctly contra-indicated, and a stimulant plan of treatment is to be followed, else sudden collapse of the system may come on. Alcohol with opium at bedtime, rest in bed, nutritive food, &c., are the measures which contain the most promise in these cases.

In those forms of cerebral hyperæmia which are sudden in their origin, and are dependent upon some surprise, mayhap of an agreeable character, denominated psychical intoxication, the two factors—the vascular and the tissue-factor—are found together. A typical case, recorded by Dr. Crichton Browne, is quoted by Dr. Handfield Jones (2nd ed., p. 159), where a youth of nervous temperament becomes temporarily deranged from the agreeable surprise of a sum of money coming to him. The condition closely resembled alcoholic intoxication. It terminated in vomiting and heavy sleep, In a couple of days the patient was quite well. In his comments on this case Dr. H. Jones says : ' Dr. Crichton Browne seems to consider that the emotion told principally on the vaso-motor nerves of the cerebral arteries, and that the symptoms resulted from hyperæmia, due to their relaxation. To me it rather seems that the cerebral excitement was primary, that the normal action of the nerve-cells of the hemisphere and mesocephale was deranged by the surprise, and that the alteration in calibre of the blood-vessels was secondary to this disturbance, just as Schrœder van der Kolk considers it to be in epilepsy. The case is most properly termed one of *psychical* intoxication, for I have no doubt the mental stimulus was exactly the equivalent of the physical ones, alcohol or opium, which produce by their direct action in the nerve-tissue toxic phenomena so very similar.' The difference of opinion here given by two such competent authorities illustrates well the difficulty of arranging the phenomena of these cases, and of determining whether alterations in the calibre of the encephalic blood-vessels precede or follow modifications in the cerebral cells. Whichever comes first they are almost synchronous—certainly they are cotemporaneous.

This consideration of the several forms of cerebral hyperæmia will naturally lead up to the question of the thera-

peutic measures which are to be employed in the treatment
of this pathological condition. It is at once obvious that
each case will demand a line of treatment adapted to its own
individual necessities. In every case the two factors of the ·
vascular element and the local tissue element must be borne
in mind. In the treatment of the first factor it will be neces-
sary to carefully calculate the proportion of each of the two
elements, and then to apportion the amount of vascular de-
pressant which may be necessary to calm the circulation.
For this end antimony, aconite, calabar bean, or veratrium
viride may be resorted to, as typical vascular depressants.
Then will come the consideration of the other element—viz.,
the excitement in the cerebral cells. This will require its
more special remedy in the form of some neurotic which di-
minishes cerebral activity, such as opium, hydrate of chloral,
or bromide of potassium. These remedies may be given alone
or combined with each other, according to the exigencies of
each case. Opium has a very decided effect upon the nervous
system, briefly exciting and then lowering the activity of
nerve-cells. Consequently, in conditions of vascular excite-
ment with cerebral hyperæmia, it becomes desirable to com-
bine opium with direct depressants. In other cases again,
where there is great exhaustion, it is well to give it along
with alcohol and nutritive material. But opium exercises only
a slight effect upon the circulation generally, and its chief
action is upon the nerve-cells. In chloral hydrate the two
factors of action—one upon the nerve-cells and the other
upon the circulation—are differently proportioned, and chloral
acts pronouncedly upon the vascular system, lowering the
heart's action and lessening blood pressure very markedly ;
consequently it is indicated where there is much vascular ex-
citement. It may be given alone or with opium, according
to the amount of cerebral excitement present. Where there
is high excitement in cerebral hyperæmia it may be profitably
combined with bromide of potassium, alike in the delirium
of acute disease in children and in the more persistent
stages of exaltation in the insane adult. The effect of bro-
mide of potassium is felt chiefly in the nerve-cells, whose
activity is distinctly lessened by its use. (Nothnagel.) Its

action is often almost magical in lowering excitement in the nervous centres. In cases of cerebral hyperæmia with much exhaustion, as in those who have broken down from sustained mental overwork or from alcoholic indulgence, where the pulse is fast and feeble or irregular, bromide of potassium may be combined with digitalis with advantage, as an increasing experience is demonstrating. The digitalis acts upon the vascular system, giving it tone, while the sedative effect of the bromide upon the nerve-centres controls them, and as it were steadies them. There is nothing incompatible in such union of digitalis and bromide of potassium. In other cases again of cerebral hyperæmia, especially in simple, uncomplicated forms, ergotine is found very useful in practice. Max Schuler has found it to contract the arterioles of the pia mater very distinctly. In each case it will be necessary to estimate as accurately as may be the proportion of each factor, vascular or nervous, present in each case, and then to combine the remedies accordingly. In all cases it is well to act sharply upon the bowels, especially if there exist any source of irritation within the pelvis; if any erotic feeling be present, then sulphate of magnesia, with soda or potash in decoction of aloes, pushed to the extent of three or four copious motions every day, will be found of great service. The application of cold to the head will also be found useful in most cases. The food should consist of milk and seltzer water, rice water, barley water, farinaceous foods and fruits, and it is well if they are given cold or iced.

III. We now come to the third division of cerebral hyperæmia, namely, that produced by drugs. It is well known that furious delirium, general excitement, indeed the well-recognised symptoms of cerebral hyperæmia, may be produced by vegetable products, and notably by members of the Solanaceæ. Belladonna, datura stramonium, and henbane are agents which produce acute cerebral symptoms. As to belladonna, it produces a condition closely allied to, if not identical with, that present in the case quoted from Handfield Jones above, the case of psychical intoxication. In the delirium, in the acceleration of the pulse, and in other respects, belladonna poisoning is almost identical with it. This delirious excite-

ment, Jones thinks there can be little question, depends upon
arterial dilatation and increased afflux of blood. John Harley
thinks the sleeplessness of atropine poisoning due to the
cerebral vascularity induced thereby. Curiously, too, the
periods of excitement in the general paralysis of the insane,
where there is great cerebral and vascular excitement com-
bined, are controlled by calabar bean, which is the physiolo-
gical antagonist of belladonna. Some exact observations as
to the effects of belladonna upon the vessels of the pia
mater are much called for, and it is the writer's intention at
an early period to make a series of such observations.

As to the excitement produced by alcoholic intoxication
and the physical effects of this agent upon the cerebral ves-
sels, there is much agreement of opinion, and Dogiel found
that during the earlier periods of alcoholic intoxication, ere
excitement had given way to coma, the carotid arteries were
dilated, showing the afflux of arterial blood to the brain.
The effect of opium, like that of alcohol, is to dilate the
vessels of the encephalon first, corresponding to the period of
excitement, and then to produce contraction, corresponding
to the period of sleep—of inaction. Nitrite of amyl, accord-
ing to Max Schuler, produces dilatation of the intra-cranial
arterioles.

The effect of quinine in full doses is to produce cincho-
nism, a condition marked by noises in the ears, especially the
ringing of bells, flashes of light in the eyes, delirium of an
active character, wakefulness, suffusion of the eyes, redness
of the conjunctiva, and sense of distension in the head,
relieved usually by epistaxis, with throbbing of the carotid
and temporal arteries. The identity of this condition with
that previously described as the second form of cerebral hy-
peræmia is patent to all. The effect of quinine upon the
circulation is a disputed matter, Ackermann asserting that
it raises the blood pressure, while Briquet denies this, stating
that the blood pressure is lowered in proportion to the dose
of quinine. The experiments of the latter have placed be-
yond doubt the effect of quinine in producing increased
encephalic vascularity to the extent of true meningitis; ne-
vertheless, Hammond performed further experiments, espe-

cially upon himself, and corroborated the views of Briquet. After full doses of quinine he not only noted his subjective phenomena, but got his friend Dr. Roosa to examine with the ophthalmoscope his retina while under the influence of the drug. The increase in vascularity was pronounced, and an examination of the ears showed injection of the mallei, so that the increase in cerebral vascularity was demonstrated at those points where it comes within our ken. These experiments of Hammond's upon himself were accompanied by observations upon dogs, which showed conclusively the influence of quinine in causing hyperæmia and congestion of the intra-cranial circulation. ('New York Psychological and Medico-legal Journal,' October, 1874.)

The subject of the effect of drugs upon the encephalic circulation is an attractive one, not only physiologically but therapeutically, and, if space permitted, the subject might be pursued ; at present, however, this is impossible. Enough is known to indicate to us pretty clearly the means by which we can increase the vascularity in conditions of cerebral anæmia, just as we know of agents by which we can affect conditions of cerebral hyperæmia. In the latter, as in the former state, there are two factors in action, the vascular and the nervous, and in all cases in practice it is worth while to bear this in mind, especially in the selection of the remedial agents.

A NEW PROCESS FOR EXAMINING THE STRUCTURE OF THE BRAIN. WITH A REVIEW OF SOME POINTS IN THE HISTOLOGY OF THE CERE-BELLUM.

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THE methods usually adopted in the microscopical examina-tion of the brain have all proved in my hands more or less unsatisfactory. I find that when thin sections of hardened brain are cut and stained, the dye does not sufficiently dif-ferentiate the various structures so as to render their form and arrangement obvious, while in teased preparations the shape of the cells, the connection of their processes, and the fibres of the brain are just as likely to be torn to pieces as to be separated from the substance which surrounds them.

The plan which I am about to describe will, I think, be found to overcome, to a certain degree, several of these de-fects. The dye which I employ causes the nuclei to appear black; the cells and their processes are rendered dark purple, while the rest of the section is of a faint purplish blue colour, so that the processes and fibres are rendered by this means extremely distinct, and may often be readily traced to dis-tances of a quarter or half an inch, and in some cases even to greater lengths.

For the sake of clearness of description I will divide my process into several stages :—

I. The first stage consists in making slices of brain, which should be made from the organ as it is obtained from the post-mortem room, neither hardened nor altered in any way by reagents. The sections should be cut as thin as practi-cable, but slices of one-eighth of an inch in thickness will

not be found too thick for the subsequent treatment. I find the following a convenient mode of making such sections. A large brush is to be fixed to the back of the left ring-finger by means of two elastic bands; the operator then holding a piece of brain in the left hand, slices it with a large knife kept constantly wetted with spirit by means of the brush. A large amputating knife answers well for this purpose. The sections should be cut in the direction from the operator. As they are made they should be wiped off the blade with the brush, and allowed to fall into a vessel containing about half a pint of water.

The fixing of the brush in the position described will be found to give very important assistance. The piece of brain, as freshly taken from the subject, is difficult to seize and to hold in one position while making the slices as described, and the attempt to hold the brush and the brain at the same time will be found to be a work of difficulty; and as it is desirable to take several sections from the same place, the piece must not be allowed to slip from the operator's grasp, as it would be very difficult to readjust it.

II. The next stage is to subject the sections to the action of the dye. The water in which the sections were placed is to be poured off, until there remains only just enough to cover them. To this there is to be added an equal quantity of a one per cent. solution of the dye, so that in fact the sections will be now in a solution containing half per cent. of the dyeing material.

The Dye.—The material that I have found most satisfactory in its effects is called aniline blue black. It can be obtained from Messrs. Hopkins and Williams, Cross Street, Hatton Garden. In the dry state this material is a blackish powder, not unlike gunpowder in appearance. It is very soluble in water, to which it imparts an intense purple colour. Its chemical composition is, I believe, not exactly known. I regard the use of this dye as an essential part of the process. I have experimented with some thirty or forty other dyes, but have had no results at all equal to those obtained by aniline black. It is also useful for sections made after the usual process of hardening, &c.

The sections should be allowed to remain in the dye for about twelve hours; twenty-four or even thirty-six hours' immersion does not, however, often injure them. The next step is to pour off the dye, and add clear water until all the colouring fluid has been washed away. The stained sections may be poured, with the water they are in, into a large shallow basin, and each slice floated into the middle of a glass slide, and removed carefully from the water and allowed to drain. As each section in this stage should be marked, I can recommend for the purpose a very convenient slide, invented by Mr. Hicks, and described by him in 'Quart. Journ. Microsc. Science.' In this slide the portion on which the label is usually gummed is of ground-glass, and will receive a pencil-mark without danger of its being obliterated.

III. The next step in the process is to dry the stained slices upon the glasses. They should be placed in an airy and dry situation, but not as a rule, subjected to heat, but to a current of air. In certain states of the atmosphere, however, they may require the assistance of a very moderate degree of artificial heat. Or should the sections be very thick, a slight amount of dry heat is required so as to anticipate any liability to putrification before desiccation has had time to occur. When the sections are dry it will be found that they are firmly adherent to the glass, and that they, as a rule, show no tendency to crack, except just at the edges. Should it be desirable to obtain a good view of the edge of any section, a slice of unstained brain may be placed over the stained piece so as to overlap the edge, which virtually removes the edge it is desired to see from its external position, and renders it far less liable to crack.

IV. When the sections are properly dried they will be found to be of the consistence of somewhat dry cheese, and rather uneven in thickness. The next step is to bring them to a condition suitable for microscopical examination, by reducing them to the necessary thickness. The sections, before they are reduced, may be described as consisting of three layers— an upper, middle, and lower, the latter being in contact with the glass. It is obvious that the upper and the lower, which were the parts in immediate contact with the dye, will be

more deeply stained than the central layer, which in fact
remains white. In reducing the thickness of the sections
by paring, it is clear that if the upper layer is removed but
one of the deeply-stained layers will remain. The object
to be gained by the paring is to reduce the section to the
lower stained layer only. The unstained or intermediate
layer may be removed as completely as practicable without
endangering the deeper blue layer ; but if any unstained sub-
stance be left it is of not much consequence, as the process
of clearing renders it invisible. The operation of paring
can readily be performed with a razor, but it can be much
more efficiently and quickly accomplished by means of an
instrument that I have devised for the purpose. This instru-
ment is a kind of plane, and is a modification of an ordinary
carpenter's two-inch skew rabbet-plane, but with which I ope-
rate in the reverse position, that is, with the cutting edge
uppermost. The modification consists in screwing to the sides
of the plane two slips of iron, having perfectly true and level
edges, which are also adjustable by set screws, and can be raised
just above the level of the cutting blade of the plane. They
thus form guides, one on either side of the knife, by means
of which the thickness of the section is regulated. In using
the plane, each end of the glass side is pressed tightly down
on the iron on either side, so that the slide bridges over the
interval between the guides. The dried slice of brain being
on its under surface, by passing the slide along the guides
from front to back all the projecting portions of the tissue
come in contact with the knife, and are pared off until the
whole is reduced to the requisite thickness, this thickness
being regulated at will by raising or depressing the side guides.

V. The preparation is now to be cleared by means of
damar varnish or Canada balsam. The intervention of oil
of cloves is not required. The section may be examined
at once with a low power, and, if found satisfactory, a cover
glass should be placed over it.

Sections prepared as above may be made of almost any
size. I have some which cover an extent of glass equal to
three or four square inches. By employing a very long knife
sections extending from the medulla oblongata through the

pons varolii into the crus cerebri, for instance, can be readily prepared; and in many of my finished preparations fibres can be traced to extraordinary distances.

I have now treated the brains of a considerable number of animals by this method, and have also made a few preparations of the spinal cord and sympathetic and spinal ganglia of the larger domestic animals, and always with more or less satisfactory results. I find that the larger and older the animal is, the better are the preparations which are obtained. In the smaller animals the brain is softer and much more difficult to cut, and it is apt to break down in the dye into a ropy tenacious mass, showing no trace of the outline it previously presented. The brains of young and small animals become also more brittle when dried, and are liable to crack or contract in the process of drying. In spite of these difficulties, however, with care sections of even fœtal brains may be prepared. Of all brains, that of the human adult is the one for which my process is most suited, and especially, it has seemed to me, in cases in which the temperature has been elevated for some time previous to death. The process is inapplicable to brains which have been hardened by any reagent, as they will crack or become brittle in the drying; nor is it suitable for brains which have been in any fluid capable of crystallisation; for crystals form during the desiccation and spoil the preparations. If sections cannot be made as soon as the brain is removed from the skull, or at latest on the day following, the best fluid to place it in to preserve it is a strong solution of ammonium acetate of a sp. gr. of about 1·040. This solution does not harden the brain to any marked degree, nor does it crystallise out during desiccation, nor by its deliquescent properties does it prevent the brain from drying. The brain, indeed, may be preserved in this solution for many weeks in a suitable state for the process. It should, however, be cut into pieces not larger than walnuts before being placed in the fluid, and should be soaked in water for several hours before the sections are made, otherwise it will be found that the brain has for some reason become much more adhesive, and sections when made cannot be removed from the knife without laceration.

Part II.

By means of the process of which I have now given the principal details, I have carefully examined many brains, with the view of testing various views which have been published at different times in connection with the Histology of the Brain and Nervous Centres. I propose here to give some of the results which my study has afforded me, and in the present communication I intend to confine my observations to the structure of the cerebellum; since my mode of investigation enables me to confirm some observations made originally by Dr. Obersteiner [1] concerning it, which, so far as I am aware, have not yet met with substantiation, and which do not even appear to be accepted; if I may judge from the fact that though Dr. Meynert, writing in ' Stricker's Comparative and Human Histology,' alludes to several points mentioned in the paper quoted, yet omits all mention of those points to which I am about to allude.

Dr. Obersteiner, in speaking of the pure grey or outer layer of the cerebellum, says :—' The neuroglia of this layer is scattered over with round and elongated nuclei of a diameter of 0·007 mil. The latter scarcely present any cell around them, and probably belong to the connective tissue. A clear border surrounds the round nuclei, which is either round or angularly drawn out. It is these nerve cells with processes which unite themselves to the end branches of Purkinje's cells.' I find that if in a section made perpendicularly to the surface of the cerebellum, and at right angles to the lamellæ, one of Purkinje's cells be brought into view under a magnifying power of about 600 diameters and its peripheral process traced out, the following appearances will be observed. There is a slight variation in the detail, according as the cell is situated at the top or side of the lamella or at the bottom of the sulci, but the general arrangement is as follows :—(Fig. 1). Each cell gives off in

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[1] Beiträge zur Kenntniss vom Bau der Kleinhirnrinde.   Sitzungsbericht d.K.K. Acad : der : Wissenschaft, Band. LX. Heft. I.   Wein.

a direction toward the surface one primary trunk. This soon divides into two secondary trunks which pass to the right and left in a direction parallel to the surface, and in their progress send off at right angles numerous branches, which also are directed towards the surface or towards the pia mater; and, finally, the secondary branches, having gradually become smaller and smaller in their course, terminate themselves by turning, like the smaller processes, towards the surface.

At each point of division there is to be observed a small triangular swelling, which might aptly be compared to a small blot, such as might occur when a line of ink is drawn across another which is still wet. Various opinions have been broached as to the nature of this swelling. Dr. Meynert seems to have considered it due to a loose hyaline sheath which he describes as investing the cells, and as prolonged *a short distance on the larger processes.* Dr. Obersteiner, on the other hand, appears to think it due to the passage from one branch to another of fibres which do not pass back to the cells of Purkinge, but appear, as it were, to form a direct outer communication between the branches. I have been able to see distinctly this triangular enlargement at the union of some of the finest branches, such as are described below as connected with small cells in the pure grey layer, and which cannot, I think, be bundles of fibres, but must be single fibres. If such is the case, the enlargement cannot be produced in the mode supposed by Dr. Obersteiner. On the other hand, if this appearance is due to a sheath, then such hyaline investment must extend to the very finest fibres, and cannot cease at the point mentioned by Dr. Meynert.

The fibres running outwards may be seen to divide and subdivide into very fine branches, dividing not three or four times only, as might be supposed from the drawings given in the text-books or by Dr. Obersteiner, but much more frequently. I have observed one branch to divide more than twenty-five times.

The more distant branches are given off at very acute angles, and the fibres, after a very short distance, run a

nearly or quite parallel course. The division is not strictly dichotomous; for in many instances small branches are given off from the sides of larger ones, though more usually two branches of equal size result from the division of one. Dichotomous division prevails more as the fibres become smaller. The branches, having thus become finer and finer, are ultimately lost to view, or terminate in the remarkable manner described by Dr. Obersteiner (Op. cit.).

In my preparations the union of the fibres with the protoplasm of the cells, in the pure grey layer, is plain and unmistakeable, and resembles very closely the appearance described by Dr. Obersteiner, though some differences may be noted. I think it is probable that these are due to the mode of preparation of the object, and I believe that my method shows the structure to greater advantage than did the process at his command. Certainly I am able to see the structure, and the artist has been able to figure it much more decisively than Dr. Obersteiner has ventured to show it. (Fig. 2.) The fine fibres, on approaching the cells in which they are about to end, are seen in my preparations to enlarge and assume the character of the protoplasm of the cell, into which they pass without line of demarcation. Dr. Obersteiner, however, in his drawing, depicts the cell as a round ball, into which, in one sketch, a fine filament is seen to pass, and only into its proximity in his other sketch. Probably this appearance, as given by him, was due to the shrinking of the tissue in the process of hardening, for most of the cells seen in my preparations are not round, but, as Dr. Meynert describes them, triangular or pastille-shaped. Again, there is no clear space around the cell in my preparations, which in Dr. Obersteiner's drawing appears so wide as to be nearly equal to half of the diameter of the cell itself.

These small cells are not only seen to be provided with a process of union connecting them to the fibres in the manner described, but also to give off others three or four in number, which are short, but which divide and subdivide until they are finally lost in the reticulo-molecular ground substance of

the part.   The 'processes of union,' which are connected
with Purkinje's cells, are usually directed toward that layer
of cells, but this is not an invariable arrangement.   My
preparations corroborate Dr. Obersteiner's opinion that the
terminal branches of the processes are directly united to
cells, and that also side-twigs are given off in a more or less
rectangular direction, which also join cells, each fibre mak-
ing for its cell a kind of stalk.   I have, however, more fre-
quently observed the former kind of connection than the
latter.

With regard to the questions, are the processes invariably
connected with cells, and are all the cells of similar cha-
racter and appearance connected with fibres, I can give
no definite answer.   I have observed that the cells in any
given space that are unconnected with these processes are
about equal in number to the terminal fibres, which appear
to end indefinitely or appear unconnected with the cells,
though there may be a slight excess in number on the side
of the fibres.   The more intensely the preparation is stained,
the greater the number of those connections is there to be
seen.   I am, therefore, inclined to believe that it is owing to
the defective method of examination that we are still unable
to assert that the fibres uniformly terminate in cells.

With regard to the statement of Dr. Obersteiner that there
are nuclei of two kinds in the pure grey layer of Kölliker,
I may state that I believe such to be the case.   Firstly, there
are those cells which are surrounded by a protoplasmic sub-
stance, and which are the most numerous ; these are the cells
which are connected with the fibres from the cells of Pur-
kinje ; and, secondly, there are undoubtedly others around
which no protoplasm is to be seen ; these are either round or
more frequently slightly elongated, and both Dr. Obersteiner
and Dr. Meynert regard them as free nuclei belonging to the
neuroglia.   In my preparation small arteries and veins may
be detected, but no capillaries, or, if any, very few, and these
but faintly indicated ; but by taking a freshly-stained portion
of cerebellum and teasing it, the nuclei of the capillaries may
be readily detected, and will be found to resemble in appear-
ance, shape, and size the second kind of nuclei referred to

II.   × 950.
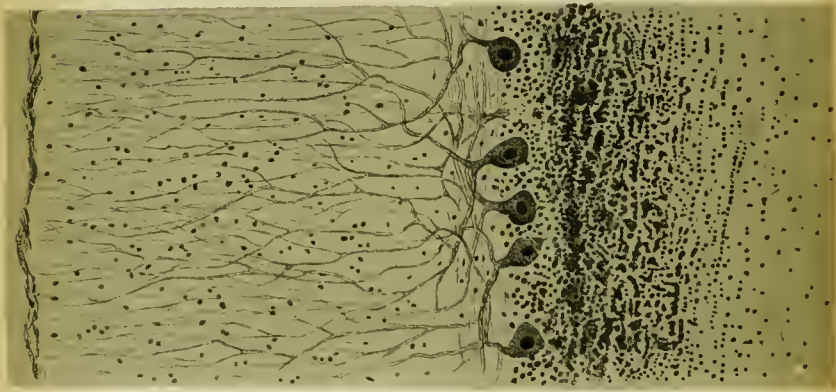
I.   × 140.

above. I am, therefore, of opinion that this kind of nucleus belongs to the capillaries, and is not an element of the neuroglia. That such is the case, I think, derives support from the fact that their numbers are about what such an origin would readily account for.

The annexed chromo-lithographs represent with great accuracy the appearance seen in the preparations, both with regard to form and to colour. Fig. 1 is magnified 140 diameters. It includes a part of every layer of the cortex of the human cerebellum.

Fig. 2 is a highly magnified view (950 diam.) of a part of the pure grey layer of the cerebellum taken at a spot rather nearer to the pia mater than to the layer of Purkinje's cells. This figure is, to a certain degree, diagrammatic. The most central process with its attached cell, which really occupied a position below and to the left of the structures represented, has been inserted in place of a fibre which did not show anything noteworthy. There was in the preparation in the left upper corner a small rent; this has not been represented; a group of six cells and nuclei taken from another part of the preparation having been inserted instead. Of the four nuclei in this group, three are elongated, and appear to belong to the vessels. The most internal one, on the contrary, is round and resembles those contained in the cells. Some clue to the existence of these free rounded nuclei may perhaps be afforded by the appearance seen at another part of the plate. In the right upper corner is a cell of which the protoplasm is so pale as scarcely to be visible, while nearer the middle of the lithograph is a nucleus nearly extruded from its cell. In the lower part of the plate are to be seen three or four unequivocal instances of union of fibres derived from Purkinje's cells with the protoplasm of small cells in the outermost layer of the adult cerebellar cortex. Such connections are, of course, faithfully copied from the preparation.

EPILEPTIFORM SEIZURES IN GENERAL PARALYSIS.

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The seizures that occur at various periods during the course of general paralysis consist of attacks varying in severity and duration, sometimes only amounting to slight vertigo, sometimes to profound loss of consciousness, and may be accompanied by simultaneous convulsive phenomena affecting the face, trunk, or extremities singly or together in various combinations, and either one side of the body more than the other, or both equally. The term 'seizure' then is used to include very different groups of symptoms (all agreeing in the sudden nature of their onset), and as no anatomical lesion has yet been localised in the brain or spinal cord that will account for these different groups, the word seizure has been qualified by expressions either intended to signify the supposed nature of the lesion, or by expressions denoting the principal symptoms, or groups of symptoms, by which the seizure is characterised.

The adjectives 'congestive' and 'apoplectiform' are, I believe, most commonly used in the first sense; 'vertiginous,' 'epileptiform,' and 'paralytic,' in the latter.

Bayle first used the word 'congestive' in connection with the apoplectiform attacks, which he considered depended upon sudden congestion of the pia mater and of the brain. Recently, however, the term has been used by authors,

chiefly the French, to express the mode of origin of the epileptiform as well as the apoplectiform attacks of the general paralytic, which have been considered dependent upon sudden congestions in the brain, especially in the medulla oblongata. It is extremely difficult to draw the line between epileptiform and apoplectiform seizures, for in both there may be impairment or loss of consciousness, of sensibility and reflex action, and in both, subsequent to the attack, there may be loss of power in various parts of the body for various periods.

In the following pages the adjective ' epileptiform ' is used to express such sudden attacks as are characterised by a series of more or less severe involuntary muscular movements, with or without impairment or loss of consciousness ; and ' apoplectiform ' such sudden impairment or loss of consciousness as occurs without convulsive phenomena. In either form a new paralysis of varying duration may follow the attack, or an increase of before-existing paralysis, and in certain cases in which patients, not under continuous supervision by night as well as by day are found to have become hemiplegic, or paralysed in various parts, during the time that they were not under observation, it will be difficult or impossible to determine what was the mode of origin of the attack. For this reason, in the accompanying tables I have placed the two classes in neighbouring columns, and the doubtful cases have been included with the apoplectiform seizures.

Dr. Hughlings Jackson[1] has pointed out that there are three lines along which the investigation of any case of nervous disease may be pursued. My inquiry has been mainly directed to the second of these lines, the functional affection of nerve tissue in cases of epileptiform seizures of general paralysis, and with this object I have sought for information in the valuable records of the West Riding Asylum. For permission to do so, and for courteous assistance in many

[1] 'West Riding Lunatic Asylum Medical Reports,' Vol. III. 'Investigation of Epilepsies.' ' We have—1. To find the organ damaged (localisation). 2. To find the functional affection of nerve tissue. 3. To find the alteration in nutrition.

ways, I must here acknowledge the debt I owe to Dr. Crichton Browne.

Prevalence of epileptiform seizures in cases of general paralysis.—The occurrence of these seizures is spoken of as so common as to occur in nearly every case of general paralysis. From the notes of the clinical histories of 100 general paralytics, taken consecutively, who ended their days in the West Riding Asylum, I find that this statement is certainly borne out, though not so fully as I should have expected. Of these 100 cases sixty are recorded to have been affected by marked seizures at some time while they were under observation and treatment at the Asylum, and the remaining forty either were not so affected or had only slight faintings, &c. Some of these cases, too, were admitted into the Asylum in such an advanced stage of the disease that seizures had already most probably occurred. Lastly, there may have happened unnoticed during the night in several cases seizures of a slighter nature, the effects of which had already passed off before morning, or if they occurred in the advanced stage, were masked by the general debility of the patient.

In fifty-one out of the sixty cases who had seizures, attacks of an epileptiform character are described ; in the remaining nine cases there is either simply the record of a 'stroke,' or a fuller memorandum to the effect that the patient was observed on a certain date to be hemiplegic. I have already stated my belief that some of the latter cases may have been convulsed before the paralysis took place.

Age.—In preparing some of the following tables, I have had to go over ground similar to that already occupied by Dr. Wilkie Burman, the results of whose inquiries are contained in the first volume of these Reports. Thinking it might be interesting to compare on some points the statistics of the Devon County Asylum and those of the West Riding Asylum, I have arranged as far as possible the cases I have collected at the latter place in a similar manner to Dr. Burman's cases.

On comparing the ages on admission of the first group with those of the second, it does not appear that the liability

of a patient to seizures increases in proportion with his age when attacked, or in other words that the greater the age of a patient when attacked with general paralysis the greater his chance of a seizure during its course; for of fifty cases in which epileptiform seizures occurred, twenty-eight cases, or about 56 per cent., were less than forty years of age when attacked by general paralysis; while of forty cases in whose histories no seizures are recorded, only sixteen were less than forty years of age when attacked (40 per cent.). Of the first mentioned class again, sixteen were between forty and fifty years of age (32 per cent.), while of the latter class twenty patients out of forty (50 per cent.) belonged to this decade. Finally, the average age of the total number of the last class is more by one year than that of the first, the difference between the average ages of the female patients being especially noticeable.

TABLE I.

Age on admission	All cases			Reported to have had seizures						Not reported to have had seizures		
				Epileptiform			Apoplectiform					
Years	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total
From 20 to 30	4	—	4	1	—	1	—	—	—	3	—	3
„ 30 to 40	34	9	43	20	7	27	2	1	3	12	1	13
„ 40 to 50	33	8	41	14	2	16	4	1	5	15	5	20
„ 50 to 60	9	2	11	6	—	6	1	—	1	2	2	4
Doubtful	—	1	1	—	1	1	—	—	—	—	—	—
Totals	80	20	100	41	10	51	7	2	9	32	8	40
Average age.				40·	38	40·4	42·77–35		40	40·43–45·37		41·42

Average age of 59 cases reported to have had seizures, 40·14.
Average age of 40 cases not reported to have had seizures, 41·42.

Sex.—The proportion of males to females was found at the Devon Asylum to be 4 to 1, or a little over. This proportion is, I also find in the 100 cases I have taken, to be observed both in those patients reported to have had seizures, and those not reported to have had them. Table I. shows the distribution of the sexes under various ages.

Duration of the disease.—It will be seen from the accompanying table that the average duration of the disease in the

total number of cases was 21·59 months; but while the average duration of the disease in patients who had epileptiform seizures was 22·3 months, in those supposed to have passed through the various stages of the disease without any seizure was only 19·9 months. The average duration in patients who had only apoplectiform seizures was 27·1 months, the highest of the three classes. (Dr. Burman found the average duration in thirty-two cases to be one year and nine months.) With regard to the influence of *sex* upon the duration of the disease, the table shows that in all three classes the duration was longer in the case of female patients, and the difference in the averages of the total numbers amounted to 5·2 months. (Dr. Burman found the difference to amount to twelve months between the total duration in thirty-two males and that in six females.)

TABLE II.

Total duration of the disease.	Reported to have had seizures						Not reported to have had seizures			All cases. Grand Total		
	Epileptiform, &c.			Apoplectiform								
Months	M.	F.	Total	M.	F.	Total	M.	F.	Total.	M.	F.	Total
Under 3	0	0	0	0	0	0	3	0	3	3	0	3
From 3 to 6	2	1	3	0	0	0	3	0	3	5	1	6
,, 6 to 9	4	0	4	1	0	1	5	1	6	10	1	11
,, 9 to 12	3	0	3	0	0	0	0	1	1	3	1	4
,, 12 to 24	15	4	19	2	0	2	12	2	14	29	6	35
,, 24 to 36	10	3	13	3	1	4	4	1	5	17	5	22
,, 36 to 48	4	2	6	1	1	2	3	3	6	8	6	14
Above 48	3	0	3	0	0	0	2	0	2	5	0	5
	41	10	51	7	2	9	32	8	40	80	20	100
Average dura-⎱tion in months ⎰	22	23	22·32	24	38	27·1	18·3	26·5	19·9	20·7	25·9	21·59

Relationship between age and total duration.—Table III. shows that in the total number of cases the duration of the disease is very nearly the same at whatever age the patient is attacked. Where epileptiform seizures occurred, the duration of the disease diminished in proportion with the increase of the age at which patient was attacked. Thus in nine patients who were attacked before they reached the age of thirty-five the total average duration of life was 28·1 months, while six patients who were more than 50 years of age when attacked only reached an average of **16·16**

months. There is no such gradual shortening of the term of life observable in patients who either had apoplectiform seizures, or were not reported to have had any at all.

TABLE III.

Relationship between Age and total duration	Reported to have had seizures				Not reported to have had seizures		Totals of all cases	
	Epileptiform		Apoplectiform					
Years of Age.	Cases	Avge. Ms.	Cases	Avg.Ms.	Cases	Avg. Ms.	Cases	Avg.Months
Before 35	9	28·1	2	26·5	8	14·6	19	22·2
From 35 to 40	19	23·3	1	13·0	8	22·3	28	22·7
„ 40 to 45	8	20·6	3	25·0	9	21·0	20	21·4
„ 45 to 50	8	19·6	2	32·0	11	20·0	21	21·0
Above 50	6	16·16	1	39·0	4	23·5	11	20·9
Of Unknown Age	1	?	0	0	0	0	1	?
	51	?	9	27·1	40	19·9	100	?
Average Nos. .	50	22.32					99	21·5

Residence in Asylum at time of death.—The average duration of Asylum residence in fifty-one cases of general paralysis in which epileptiform seizures occurred was slightly (1·5 months) longer than in the case of forty patients who had no seizures. Grouping with the former cases those patients who had apoplectiform seizures, the difference is greater, amounting to 4·28 months. In Asylum residence, as in the total duration of the disease, the female patients have the advantage, the difference in the averages of the total numbers of each sex amounting to 3·3 months. By subtracting the average term of Asylum residence from the average total duration of the disease, the approximate duration of the attack previous to the admission of patients in the three classes into which the total number of general paralytics are here divided, may be arrived at. In the total number of cases this averaged 4·7 months in males, and 6·6 months in females. In the epileptiform seizure cases it averaged, males 5·61 and females 6·2 months. In those who had apoplectiform seizures only, males 2 months and females 1 month. In those who had no seizures at all, it averaged, males 4·1 months, females 8·5 months.

TABLE IV.

Residence in Asylum at time of death.	Reported to have had seizures						Not reported to have had seizures			Grand total All cases		
	Epileptiform			Apoplectiform								
	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total
Months												
Under 3	3	1	4	0	0	0	6	1	7	9	2	11
From 3 to 6	6	1	7	1	0	1	7	0	7	14	1	15
„ 6 to 9	4	0	4	0	0	0	4	1	5	8	1	9
„ 9 to 12	1	2	3	2	0	2	1	1	2	4	3	7
Years												
„ 1 to 2	16	3	19	0	0	0	6	3	9	22	6	28
„ 2 to 3	9	2	11	3	1	4	5	2	7	17	5	22
Above 3	2	1	3	1	1	2	3	0	3	6	2	8
	41	10	51	7	2	9	32	8	40	80	20	100
Average duration in months	16·39	16·8	16·47	22	37	25·3	14·2	18	14·97	16·0	19·3	16·67

Period of the disease in which epileptiform seizures occur. —It was only in a few cases that epileptiform seizures occurred earlier than the third month after the commencement of the disease. In one of these cases the patient, a female forty-five years of age, was stated to have first shown symptoms of mental disorder only four weeks before admission, and to have had a seizure within that time. Her first seizure after her admission into the Asylum took place before she had been under treatment there two months. Dr. Crichton Browne's remarks upon the recognition of mental symptoms which mark the earliest stage of brain-wasting[1], are peculiarly applicable also to the recognition of mental alteration at the commencement of general paralysis. The most common time for seizures to occur was about the fourteenth or fifteenth month after the date at which the attack was alleged to have begun, one-third of all the cases having been first affected by either epileptiform or apoplectiform seizures between the twelfth and twenty-fourth month of the disease.

[1] Clinical Lectures on Mental and Cerebral Diseases. 1. Brain Wasting. ' British Med. Journal,' April 29th, 1871.

TABLE V.

Interval between the commencement of the disease and the first seizure	Epileptiform &c.			Apoplectiform			All cases of seizure		
	M.	F.	Total	M.	F.	Total	M.	F.	Total
Under 3 months	0	1	1	1	1	2	1	2	3
From 3 to 6 ,,	4	1	5	1	0	1	5	1	6
,, 6 to 9 ,,	5	1	6	1	0	1	6	1	7
,, 9 to 12 ,,	6	2	8	0	0	0	6	2	8
,, 12 to 24 ,,	16	3	19	1	—	1	17	3	20
,, 24 to 36 ,,	5	1	6	3	—	3	8	1	9
,, 36 to 48 ,,	4	1	5	—	1	1	4	2	6
Above 48 ,,	1	0	1	—	—	--	1	—	1
	41	10	51	7	2	9	48	12	60
Average interval in months			17·6			19·0			17·81

Interval between the occurrence of the first seizure and death. —In twenty-four out of sixty cases, death occurred within a month after an attack—whether epileptiform or apoplectiform—of such a character as to have been brought under notice. Of these rapidly fatal cases twenty-three were males, that is to say, nearly half of the total number of male patients affected by seizures died within a month after the occurrence of the first pronounced attack. Whatever the cause of the longer duration of the various stages of general paralysis in female than in male patients may be, the general tendency is brought out in this as in the other tables. In several cases seizures are not recorded to have taken place until within a few days, or sometimes hours, of death; and then the epileptiform seizures appear to have been more remarkable for the long continuance and frequent repetition of the convulsions and the depth of the coma, than for the

TABLE VI.

Interval between the first seizure and death	Epileptiform, &c.			Apoplectiform			All cases of seizure		
	M.	F.	Total	M.	F.	Total	M.	F.	Total
Under 1 month	21	1	22	2	0	2	23	1	24
From 1 to 3 ,,	3	3	6	1	1	2	4	4	8
,, 3 to 6 ,,	7	1	8	3	1	4	10	2	12
,, 6 to 9 ,,	1	3	4	—	—	—	1	1	4
,, 9 to 12 ,,	3	0	3	—	—	—	3	—	3
,, 12 to 18 ,,	5	0	5	—	—	—	5	—	5
,, 18 to 24 ,,	1	1	2	1	—	1	2	1	3
Above 24 ,,	0	1	1	—	—	—	—	1	1
	41	10	51	7	2	9	48	12	60

violence of the convulsions. The longest interval was one of thirty-two months, in the case of a female patient, thirty-five years old on admission.

Age and occurrence of first seizure.—The interval between the commencement of the disease and the first occurrence of a sicsure of an epileptiform character, is shown in the following table to be very considerably longer in patients attacked before the age of thirty-five than in those attacked during any subsequent period of five years, the difference amounting to about $5\frac{1}{2}$ months. The average interval is about the same for the next three periods of five years, but becomes much shorter in patients who were over fifty years of age when symptoms of insanity were first noticed in them.

The cases of apoplectiform seizure are hardly numerous enough for comparison.

TABLE VII.

Age and occurrence of first seizure	Epileptiform &c.		Apoplectiform		All cases Grand total	
	Number of cases	Average interval	Number of cases	Average interval	Number of cases	Average interval
Before 35 years	9	23· 0	2	24·5	11	23·27
From 35 to 40 ,,	19	17· 5	1	8·0	20	17· 1
,, 40 to 45 ,,	8	17· 5	3	14·3	11	16· 6
,, 45 to 50 ,,	8	18· 0	2	24·5	10	19· 3
Above 50 ,,	6	12·16	1	22·0	7	13·57
Unknown.	1	15· 0	—	—	—	—
	51	17·6	9	19·0	60	17·81

The following are some of the conclusions that I think are justified by the above statistics :—

I. That there does not exist such a relationship between the age of a patient when attacked by general paralysis, and his liability to epileptiform seizures as to indicate that the greater his age when attacked the greater his chance of having a seizure. In other words, that the liability of a general paralytic to epileptiform seizures is not proportionate to his age when attacked by general paralysis.

II. That the total duration of general paralysis is not shortened to any great extent by the occurrence of epileptiform seizures.

III. That in patients who have epileptiform seizures the disease is likely to be of longer duration in the younger than in the older.

IV. The conclusion to be derived from this table is similar to but better based than that drawn from Table II., for while the *total duration* of the disease is calculated by the addition of the time during which the disease is conjectured by patients' friends (whose conjectures on this point are for reasons already referred to most unreliable) to have existed to the time of residence in the asylum, there can be no possible doubt as to the accuracy of the entries from which the length of asylum residence was in each case drawn out.[1]

V. and VI. That although epileptiform seizures occur most frequently towards the close of the disease, they may also occasionally happen within a short time after the commencement of the disease. Griesinger states that epileptiform seizures *never* occur until the disease is far advanced.[2]

VII. That patients attacked by general paralysis at an early age are not likely to suffer from seizures so soon as those advanced in years at the commencement of the attack.

Etiology.—There is as yet no accurate knowledge as to what part of the brain or spinal cord is in such a morbid state as to originate the characteristic convulsive phenomena of the epileptiform seizure of general paralysis on the addition of some transitory or continuing stimulus, whether centric or eccentric. Is the grey matter of the medulla oblongata to be considered in such a morbid state of irritability that either increase or decrease of its normal supply of blood through some affection of the vaso-motor system, or that direct transmission of an abnormal impression from the altered ganglion-cells of the convolutional grey matter with which it is connected, will cause the repeated explosions, of which the spasms are the visible signs? Or, again, since exactly simi-

[1] It is most satisfactory to observe that the *average time of residence of* general paralytics in the West Riding Asylum is more than 3½ months longer than what is stated by Drs. Bucknill and Tuke in the third edition of their ' Manual of Psychological Medicine ' to be the generally estimated *total duration of the disease.*

[2] 'Mental Pathology and Therapeutics,' by W. Griesinger, M.D. The New Sydenham Society's Translation, London, 1867, p. 397.

lar seizures occur in cases of grey degeneration of the spinal cord, without mental disturbance, may the spinal cord be regarded as playing an important, if not the principal, part in giving rise to these attacks?

Lastly, are we, with Dr. Hughlings Jackson, to consider epileptiform seizures as essentially dependent upon ' discharging lesions ' of the cortical substance of certain convolutions of the cerebral hemispheres?

In the case of the convulsions of epilepsy, Niemeyer[1] states that ' the excitement of the motor nerves, of which the convulsions are the exponent, may be assumed to proceed from the *medulla oblongata*, and the portion of the brain lying upon the base of the skull, and this is shown—

1. By the interruption of the function of the hemispheres, which accompanies the convulsions.   It is not probable that motor impulses proceed from the hemispheres at a time when the irritabilities of the other ganglion-cells and nerve-fibres is extinguished.

2. Because convulsions, similar to epileptic convulsions, can be excited by continuous excitement of the basilar portion of the brain by means of the induction apparatus, while no such result is obtained by a like irritation of the various parts of the hemispheres.'   Professor Ferrier's experiments upon rabbits and cats, in which he so successfully produced epilectic convulsions by applying electrodes connected with *an induction* apparatus to the surface of various parts of the hemispheres, deprive this statement of its value as a ground for the above theory,[2] or at all events for the argument that the medulla is the sole seat of the lesion.

3. Because Küssmaul and Tenner, in their experiments upon animals, could still produce convulsions of a decidedly epileptiform character, after extirpation of both hemispheres.

4. Schroeder van der Kolk has found that, in all bodies of epileptics where the disease had been of long standing, besides numerous inconstant lesions, there was always a dilata-

[1] ' Practice of Medicine.'   Translation by Drs. Humphreys and Hackley, page 354.

[2] ' West Riding Lunatic Asylum Medical Reports, London, 1871, Vol. I p. 39.

tion of the arterioles and capillaries of the medulla, with thickening of their walls.' A hyperæmic condition of the cortical matter of the hemispheres was, Ferrier [1] states, in the experiments referred to above, observed in all cases. On the other hand, Küssmaul and Tenner produced epileptiform convulsions, with loss of consciousness, by cutting off the supply of arterial blood to the brain. Hence it seems probable that morbid instability of grey matter may arise from either increase or diminution of its blood supply. Küssmaul and Tenner considered that in epileptiform convulsions sudden localised arterial anæmia might be caused by transitory spasm of the muscular fibres of the arteries, due to a temporary excitation of the vaso-motor system; and Professor Westphal points out, considering the morbid changes in the spinal cord, in the medulla, pons and crura cerebri, that have been ascertained to exist in cases of general paralysis, such an affection of the vaso-motor nerves proceeding from these parts would not be altogether opposed to pathological anatomical facts.

Is it possible that the suddenly occurring intense *hyperæmias* that occasionally affect the surface of the body in general paralytics in an advanced stage, who have undergone epileptiform seizures, ought also to be attributed to some morbid action of the vaso-motor system? In these abortive seizures (?) the face and surface of the body generally become bright red in colour; soon after their commencement, and without any convulsions having occurred, a profuse perspiration takes place, the patient's pillows and bedding becoming rapidly saturated. The temperature of the body, as taken in the axilla, is at first extremely high, but lowers as the perspiration begins to evaporate; the pulse, at first quick and rather jerky, becomes firmer; and the breathing, hurried when the attack begins, becomes more natural as it passes off. In cases that have lately come under my notice, consciousness has been but slightly if at all impaired, and no paralysis has followed the seizure.

Professor Westphal [2] calls attention to the remarkable

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[1] *Loc. cit.*

[2] Epilepti- und apoplectiforme Anfälle der paralyt. Geisteskranken. Griesinger's Archiv für Psychiatrie, &c. I. s. 337.

resemblance existing between certain epileptiform seizures of peculiar nature that he has frequently observed in general paralysis, and those induced by Dr. Brown-Séquard in guinea-pigs after injury to the spinal cord.

The fits he describes are somewhat of the following character :—The muscles of the neck and face are seized with spasms, which succeed one another at regular intervals, with short cessations, and affect the right and left sides of the body alternately. First the head turns slowly towards the right, the eyeballs are rolled towards the same side, the right eye is firmly closed, the right corner of the mouth is drawn outwards and upwards ; then immediately there begins vibration of the muscles affected by the spasms, which gradually passes into clonic twitchings, in which too the superficial muscles of the neck (of the same side) and the tongue take part ; then the eye opens, the twitchings in the orbicularis palpebrarum and in the upper muscles of the face discontinue, the eyes roll slowly towards the opposite side, the head is turned towards the left, and a series of the very same phenomena begins on this side, only, after its completion, to cross over to the right side again. Trismus is usually noticed during these attacks. Not infrequently the muscles of the extremities participate in these seizures in quite as regular a manner as those of the face, but frequently the appearances in these cases are irregular, and without distinct connection with those of the countenance of the side affected. In many instances, too, the spasms confine themselves to one side, or, after convulsions have repeatedly recurred on one side, pass over to the other ; they are also sometimes weaker on one side than on the other.

In the guinea-pigs whose spinal cords had been injured, Dr. Brown-Séquard observed that fits generally made their appearance some weeks after the injury. In these cases, if a seizure is brought on by irritation of the skin of the face in certain localities, there follow spasmodic closure of the orbicularis, drawing of the head toward the shoulder, and subsequent twitchings of the tongue, of the muscles of the face, and eye. Should the seizure be a complete one the animal immediately falls, and there follow tonic and clonic

convulsions of the muscles of the trunk and limbs (except those that are perhaps paralysed), the respiratory muscles especially taking part.

Here, at all events, the fit must be considered *reflex* in character. May, Professor Westphal asks, the above-described characteristic attacks of the paralytic be explained in a similar manner? 'I have not succeeded,' he adds, 'in producing artificially in the latter (the general paralytic) attacks similar to those described, still the subject might be worthy of a further investigation.'

Griesinger (*op. cit.*, p. 396) makes the following observation :—' The cutaneous sensibility occasionally presents a remarkable peculiarity. While it in all cases appears at the commencement to become blunted, and afterwards in certain cases almost abolished (so that the patients can endure severe pinching without experiencing pain), there occasionally occur transitory states of extreme hyperæsthesia of the cutaneous surface, in which the slightest touch excites the most extended reflex movements, convulsions of all the voluntary muscles, conditions which present the greatest similarity to the symptoms produced in animals by a poisonous dose of strychnine. In a well-marked case of this kind we were able minutely to observe this cutaneous hyperæsthesia in the hours immediately succeeding an attack of convulsions.' In the case of the patient G. P. (see case number 5), who had been having seizures very frequently, it was noticed on one occasion that, though there were no twitchings of the legs (which had been specially affected in his seizures) on first uncovering them, exposure speedily brought them on, both legs being equally involved. Westphal also mentions that in cases of unilateral paralysis, after unilateral convulsions, there occasionally exists very decided hyperæsthesia of the non-affected side, with no increase of sensibility, or an increase to a less degree on the side that has been convulsed, but does not, so far as I am aware, give any instance of the production of a seizure by an external stimulus.

If we consider the epileptiform convulsions to be due to ' discharging lesions,' there seems to be no evidence through which we may localise the grey matter to whose discharge

the convulsions are to be attributed.   That epileptiform convulsions can be produced after extirpation of the cerebral hemispheres has already been mentioned, and Schrœder van der Kolk, believing that *epileptic* seizures depend mainly on an increased afflux of arterial blood to the medulla oblongata, compares the ganglia of that region to a Leyden jar, or to the electric organ of certain fishes ; and an epileptic fit to the spark from the Leyden jar, or to the discharge of the electric organ of the electric fish, and suggests that in the intervals of the seizures the ganglia reload themselves, as it were, for a fresh explosion.

Without wishing to do more than merely suggest a few grounds upon which the convulsive seizures of general paralysis might in some cases be considered directly or indirectly dependent upon lesions of the cortical substance of the convolutions of the cerebral hemispheres, I have ventured to repeat some of the conclusions arrived at by Professor Ferrier after his experiments upon animals, and stated in the third volume of these reports.   The first is, that 'the *anterior* portions of the cerebral hemispheres are the chief centres of voluntary motion, and the active outward manifestation of intelligence.'   The second is, that 'the convolutions are separate and distinct centres, and in certain definite groups of convolutions are localised the centres for the various movements of the eyelids, the face, the mouth [and tongue], the ear, the neck, the hand, and foot.'   The fourth conclusion, and a most important one, is that, 'the proximate causes of the different epilepsies are, as Dr. Hughlings Jackson supposes, "discharging lesions" of the different centres of the cerebral hemispheres.   The affection may be limited artificially to one muscle, or group of muscles, or may be made to involve all the muscles represented in the cerebral hemispheres, with foaming at the mouth, biting of the tongue, and loss of consciousness.   When induced artificially in animals, the affection first invades the muscles most in voluntary use, in striking harmony with the clinical observations of Dr. Hughlings Jackson.'

In addition to these conclusions, arrived at after actual experiment, we have the *clinical* fact that the psychical con-

dition is much worse after epileptiform seizures, and that though a certain amount of temporary improvement may take place, the mental powers never quite regain their former level; and lastly, the *anatomical* facts, that those changes connected with the membranes and the surface of the brain that are most characteristic of general paralysis, are almost invariably more marked in the anterior regions of the brain than in the posterior. I refer to thickening and opacity of the arachnoid, adhesion of the pia mater to the summits of the gyri, increased vascularity of the cortex, &c., and to wasting of the convolutions. Microscopic appearances peculiar to the cortical substance in the anterior regions of the brain in general paralysis have not, so far as I am aware, been described.

Premonitory symptoms are not very definite. There is sometimes increased dulness noticed, and sometimes great restlessness, loss of sleep, and refusal of food.

Actual symptoms.—The epileptiform seizures of general paralysis present all the variations in intensity of affection of consciousness and of the motorial system that are observed in cases of epilepsy. Loss of consciousness may be entire or partial, or may be impaired to such a slight degree and for so short a duration that the patient neither staggers if standing up, nor loses his balance if sitting down, and can only be suspected by a sudden alteration in the expression of the patient. The spasms, usually tonic at first and clonic afterwards, vary as to locality, severity and extent, and duration. The muscles of the face may be alone affected, or those of the arm and leg also, but it is rare to find the muscles of the neck alone involved. There may be synchronous twitchings of different grand distant groups of muscles—thus those of the face in action at the same time as those of the arm, or, more rarely, of the leg. The convulsions may be confined to one side during an attack, or, although general, may be more violent upon one side than the other; or, again, there may be alternate action. In some cases I have noticed that the twitchings of the upper and lower extremities were not synchronous. It is rare for the respiratory and abdominal muscles to be attacked at all, still more rarely with any degree of

severity.   By examination of the notes of about sixty cases,
I find that it is far more common for the convulsions to be uni-
lateral than general, and that the right and left sides of the
body in the unilateral cases are affected in about equal pro-
portions.

The other symptoms bear a close resemblance to those of
epilepsy, but are generally of a slighter character.   Respira-
tion is usually far less interfered with, and tonic contraction
neither so severe or prolonged ; the pupils become more un-
equal than before the attack ; the face may either be pale or
congested, usually the former ; the pulse is rarely so irregular
as in marked cases of epilepsy.   The after-symptoms are
extremely various.   The patient is generally stupid or sleepy,
his skin hot and perspiring, his breathing rapid and often
irregular, and his muscular condition relaxed.   Clonic
spasms frequently recur at shorter or longer intervals of
greater or less severity, and the whole attack may last from
an hour or two to two or three days.   One of the most
striking after-symptoms is the occurrence of unilateral
paralysis, which follows in nearly every case of unilateral
convulsion or of general convulsion more severe on one
side than the other.   Though the loss of power is in
some cases so transient as to have passed away and left no
trace in a very few hours' time, it may last for days or even
weeks, and rarely, I might say very rarely, for months.   In
the latter cases the paralysis has all the character of that
following extensive hemorrhagic apoplexy.

As regards the temperature of the body before, during,
and after epileptiform seizures, there is much that is
interesting to be noted.

Güntz [1] states that shortly before, or at the commencement
of the convulsions in general paralysis, he found that the
temperature fell (as he had already noticed in convulsive
seizures of tetanus), that during the convulsons it rapidly
rose again in almost one-third of all his cases as early as the
first minute, frequently within the third, but occasionally
later ; the longer and more severe the spasms, the higher

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[1] 'Temperaturbeobachtungen bei tetanischer Krämpfen, &c.' · 'Allgemeiner
Zeitschrift für Psychiatrie,' 25.

and more speedy the elevation. The amount of descent noticed, either before or together with the onset of the convulsions, appears to have been trifling, only amounting to between 0·05° C. and 0·625° C. The most rapid fall was 0·1° in ten seconds; in another case it amounted to 0·05° C. in one minute and a half, and in a third to 0·1° C. in three and a half minutes.

Clouston[1] found that 'the epileptiform fits of general paralysis are always followed by a greatly increased temperature lasting for several days,' and describes one case in which the temperature *sank* for an hour or two (after an attack?), and went up in twenty-four hours 2·5°, and in thirty-six hours 6·6°, in one case after two such attacks.

Professor Westphal[2] gives full particulars of the variations of temperature ascertained during and after epileptiform seizures in nine cases of general paralysis. He found that, with two exceptions, a very considerable rise of temperature took place after every seizure. The first exception was a case where the temperature only rose to 101·48° F. after a slight seizure, apparently unaccompanied with convulsions. The second exception was loss of consciousness but no convulsions, and here the observation taken in and immediately after the seizure showed an actual fall of temperature, which stood only at 96·62°. (By a special series of observations Professor Westphal had found the normal limits of the temperature *in ano* to be between 99·14° and 100·94°.)

The rapidity of the rise of temperature varied from 2·7° in fifteen minutes in the most rapid case downwards. In another seizure the temperature rose from 101·84° F. immediately after the seizure to 104·18°, or 2·34° within forty minutes. (In a case reported by Dr. Saunders, in the Devon County Asylum Reports for 1864, the temperature rose in one hour after a seizure from 98° to 105°, and next day a still further rise to 106° was noted. The patient died

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[1] 'Observations on the Temperature of the Body of the Insane.' J. M. Sc., April, 1868.

[2] ' Einige Beobachtungen über die epileptiformen und apoplectiformen Anfälle der paralytischen Geisteskranken mit Rücksicht auf die Körperwärme.' Griesinger's Archiv für Psychiatrie, I. s. 337.

in thirty-six hours from the commencement of the attack.) In some cases it was ascertained that the rise of temperature continued one or more hours after an attack, and in one case where it stood at 101·12° immediately after an attack, an hour later it was 102·74°, thus showing a rise of 1·8°. In another the temperature rose from 100·4°, taken immediately after a seizure, to 104·9° within six hours, a rise of 4·5°. In a third case no considerable rise of temperature was shown within twelve hours after the attack, the temperature in the morning, taken very soon after the seizure, being 100·2°, and in the evening only 101·6°. Next morning, however, it had risen to 103·1°, but here there was considerable (an increasing) sopor, with symptoms of lung disease. The temperature on the second evening was 106·3°, after which time the patient rapidly sank, and was afterwards found to have pneumonic infiltration of his left lung.

The decline of temperature in cases not immediately followed by death was found to take place at various rates of speed. In one case, where a rise of 2·34° took place in forty minutes, there was a fall of 3·06° in about six hours; in another case a fall of 1·08° occurred in three hours. In other cases a high temperature was observed to continue for some days, even up to fourteen days (with remissions and exacerbations) in one case.

The above temperatures were taken with certain precautions in the rectum, and must be compared with the normal range in that region, which, as already stated, was found to be between 99·14° and 100·94°.

In looking for a cause for the rise of temperature, Professor Westphal divides his cases into three classes. In the first he puts those cases in which he considers the rise to be undoubtedly based upon respiratory affections—pneumonia, bronchitis, œdema, &c.; in the second class there was the probability that it depended upon these diseases, their existence to a greater or less degree having been ascertained; and, lastly, in the third class are cases from which the possibility of such causes could not be excluded. He is of opinion that the frequency of inflammatory lung affections, after seizures, may, in a great measure, be accounted for by

the supposition that the passage of fluids from the mouth into the trachea and bronchi being rendered easy by the suspension of sensibility and failure of reflex action, foreign substances would find their way into the lungs.

He argues that the elevation of temperature does not depend upon the convulsions, as a similar rise has been observed in seizures where there was loss of consciousness without the occurrence of any convulsions.

Very different are these results of observation from those of Dr. Clouston [1] in epileptics.

The latter found that the immediate effect of an epileptic fit was to depress the temperature in certain circumstances as much as ·75° per hour for three hours. Though such is the immediate result, the after-effect is to raise the temperature 1·2° in from one to five hours from the time of taking the last fit, where two fits have occurred during the day. He adds that a rise of 3°, though it may occur where the attacks have produced a stupefied, confused state, lasting for many hours, is very rare, and always falls again within twelve hours.

As Westphal points out, there is a greater immunity from secondary lung affections in epileptics than in general paralytics after attacks; and this may be due to the usually much shorter duration of insensibility in the former, or to their far better state of nutrition.

In some cases of hemiplegia following convulsions I have found a considerable difference as regards temperature between the two sides of the body. Two cases that came under my notice at the West Riding Asylum, who had hemiplegia of the left side after severe epileptiform convulsions affecting almost solely that side, showed considerable daily variations. In both there was anæsthesia and congestion, followed by œdema, with formation of large watery blebs on the left hand and foot, and in both convulsions recurred, again affecting the left side. In these cases sometimes the temperature in the axilla of the paralysed side was greater than that of the non-affected side, and sometimes the reverse.

[1] *Loc. cit.*

The difference seldom amounted to more than 1° or 1½° F. and was due to the extremely high temperature attained, sometimes so suddenly, in many cases of general paralysis, in the interval between severe epileptiform or apoplectiform seizures; and death, if closely succeeding them, is a peculiarity not confined to this disease.

It has been observed in cases of epilepsy shortly before death during the 'status epilepticus,' and is also stated to occur in tetanus by Wunderlich,[1] who considers that 'any considerable elevation of temperature, in patients suffering from neuroses, when no particular reason can be assigned for the fever, is developed, affords the worst possible prognosis.' He also thinks that the frequent occurrence of such an elevation towards the close of cases of tissue changes of the brain and upper part of the spinal cord 'appear to show that there are, apparently, moderating centres or apparatus in the brain, the paralysis of which is succeeded by a morbidly increased action of the processes which produce warmth.'

Pulse.—Both during and after the seizures, the pulse seems to have some relationship with the temperature of the body, and sometimes reaches a very high rate, 160 to 180. After seizures of average severity it ranges between 112 and 128. Still it presents numerous irregularities, occasionally numbering far less than the normal (55 to 75); and this usually happens where there is prolonged stupor, hardly amounting to coma, after frequent seizures.

According to Voisin,[2] in general paralytics after epileptiform seizures the pulse-tracings show a considerable elevation of the line of ascension, excessive dicrotism, &c., and he compares these tracings with some taken after seizures in epileptics, in order to show their resemblance between the two classes of seizure. On reference to Mr. Thompson's tracings of the pulse in cases of epilepsy, taken both during and after a fit, I am unable to discover either of these characteristics.

While concluding this most imperfect description of

[1] 'Medical Thermometry.'   Syd. Soc.'s Trans. p. 425.
[2] De la paralysie générale.   Conférences cliniques, &c.   Extrait de leçons recueillies par M. Cornillon.   Union Méd. No. 85, p. 87.

phenomena, not the least interesting or important that occur in the course of general paralysis, I must acknowledge the assistance I have derived from certain papers and references most courteously presented to me by Professor Westphal. My thanks are also due to Dr. Major, who kindly supplied me with notes of several of the following cases, taken from the records of the West Riding Asylum.

Cases Illustrating Varieties of Epileptiform Seizures of General Paralysis.

I. Cases in which one side of the body was alone or principally affected.

(*a*) Unilateral convulsions not followed by marked hemiplegia.

Case 1.—T. W. (M.), æt. 38, admitted October 7, 1872. Previous duration, six months. *General condition:* Advanced in second stage of disease. April 2, 1874.—Last evening was seized with epileptiform convulsions. When visited immediately after was quite comatose, eyes wide open, pupils perfectly insensible to light, left pupil larger, breathing low and stertorous. The convulsive movements, which resembled those of a mild epileptic fit, commenced by the eyeballs rolling upwards and to the right side; after nearly a minute the right corner of the mouth began to twitch, and the skin over the neck and clavicle, the spasms being apparently confined to the depressor anguli oris and the platysma myoides. After a short interval the right sterno-mastoid began to be affected, and the head turned gradually to the right. Simultaneously, the muscles of the right arm became involved, and convulsive movement gradually extended itself over the whole of right side and the lower extremities, both of which were involved, the left arm remaining placid. Convulsions continued at intervals during the night, and patient died at 10.30 A.M. this morning.

Case 2.—P. F. (M.), æt. 43, joiner, admitted April 4, 1872. Family history of insanity. Head several times injured through falls from scaffolding. *General condition:* Beginning of second stage of disease. April 1, 1874.—Was suddenly seized with violent shiverings; became pale and cold, and afterwards vomited twice. Respirations 46, pulse 136. April 8, 1874.—Slightly better. Respirations 28, pulse 100. April 10.—Worse. Respirations 44, pulse 140. Bathed in perspiration. April 17.—Pulse 144, temperature 103·5°, respirations 50. Dulness on left side of chest. Eyes, especially the right one, rigidly closed. Strong muscular twitchings of left arm and leg. April 19, 1874.—Died at 2.40 P.M. In the morning, pulse 156, respirations 48, temperature 105·8°.

Case 3.—P. G. K. (M.), æt. 50, admitted January 6, 1873. Previous duration three years. December 20, 1873.—Had a slight stroke this morning, but the effects are already passing off. Patient now moves about in an im-

patient way, and as if pushing some one from him. Has occasional muscular twitchings all over the body, especially affecting the arms. December 22, 1873.—Has violent twitchings upwards of *left* corner of mouth. *Left* eye spasmodically closed. Abdominal muscles and muscles of legs constantly twitching. Had an inhalation of nitrate of amyl, twitchings decidedly quicker, but not quite so strong. December 27, 1873.—Contraction has entirely disappeared. Patient is conscious, and except that his speech is thicker, is in same state as before the attack. January 30, 1874.—Shortly after six o'clock this morning was seized with an attack strongly like the last. The head was turned to the right, all the muscles of the face twitched, but especially those of the left. Twitchings in the trunk very slight. Coma much more profound than before. Reflex action in right leg somewhat diminished, eyes turned to right. February 1, 1874.—Muscular movements are not so extensive or severe, and now limited to an opening and shutting of the mouth and blinking of right eye. Pulse 96. February 2, 1874.— Condition appeared better during the day, the muscular twitchings not being so severe. At night, however, they became worse, and patient rapidly sank.

Case 4.—M. F. (M.), æt. 37, admitted May 27, 1870. Previous history: Is reported to have had a slight paralytic seizure two years ago, affecting the *left* side. Memory after that time considerably impaired. *General condition:* Far advanced in second stage. November 23, 1870.—Has had two attacks of epileptiform type since admission, one some weeks ago, and the other last night. March 14, 1871.—Has spasmodic twitchings of the *left* leg. Is in the last stage of the disease. April 14, 1871.—Has this morning spasmodic twitchings of the left side of the face and left leg, the left arm lying placid by the side. Right pupil larger than the left. The spasm occasionally first attacks the face, then leaves and affects the leg. Right leg appears a little tremulous. May 5, 1871.—Twitchings on the *left* side began yesterday again. Is very feeble. May 6, 1871.—Twitchings of the left side have continued; they were stronger in the night than yesterday or to-day. Died May 16, 1871.

Case 5.—G. P. (M.), æt. 35, admitted October 6, 1870. Previously ill five months. Is reported to have had an epileptic attack seven years ago, and to have never been himself since. *General condition:* Is in the second stage. Muscles of face constantly twitching. April 25, 1871.—Has spasmodic twitchings of the right leg. July 14, 1871.—Has twitching of the right side, especially of the leg. These were noticed at six A.M. this morning, when patient was in a very drowsy, heavy state, and have continued until now, 10.15 P.M. July 15, 1871.—Twitchings continue, but patient, though still drowsy, takes his food well. July 16, 1871.—This morning on first uncovering the inferior extremities no twitchings were observed, but exposure soon brought them on, and in both limbs. July 17, 1871.—No twitchings observed to-day. August 29, 1871.—Yesterday at 11.30 A.M. patient had a pseudo-apoplectic seizure. He fell from his chair suddenly, and when seen by the medical officer very shortly afterwards was quite comatose, breathing heavily and with slight stertor. At 1 P.M. remained in same state. At 6 P.M. slightly more conscious, and more sensible to impressions. Temperature very high; pulse 152 per minute. This morning is still semi-comatose. Pulse 138, weak. Can swallow, but only with

difficulty. Patient lingered on in an extremely feeble state until October 2, 1871.

CASE 6.—H. B. (M.), æt. 34, admitted April 26, 1869. Has been ill for about two months. At commencement of second stage. January 4, 1871.— The disease is now rapidly progressing; patient is quite demented, and during the last few days has shown signs of paralysis of the *right* side, towards which side he drops. February 13, 1871.—Had a series of epileptiform seizures during the night. Has been completely unconscious. February 14, 1871.—Seems rather more conscious than yesterday, when he had several returns of the epileptiform attacks. To-day has rapid and simultaneous twitchings of the right arm and leg. In the arm the twitchings seem almost confined to the supinator longus, in the leg to the extensor longus pollicis and the peronei, and in the thigh to the quadriceps. May 30, 1871.—Had a severe epileptiform attack yesterday about two P.M. The head was drawn slightly over the right shoulder, but the eyes were not rolled up. The face was not distorted, nor the teeth clenched as in epilepsy. Mouth was slightly open. Was sick twice during the attack. Skin was hot and perspiring. There was intense subsultus tendinum, rendering it impossible to feel the pulse. He expired soon after the attack.

CASE 7.—D. T. (M.), æt. 38, admitted August 31, 1870, reported to have had epileptiform seizures. *General condition:* Advanced in second stage. October 20, 1871.—Much demented; has lost his exalted ideas. Has much tremor of lips and tongue. Gait very unsteady. January 1, 1872.—Had a seizure yesterday. He suddenly fell down in an unconscious state, and remained so half an hour. Was generally convulsed, but more particularly on the right side. Right pupil twice the size of the left. Is now conscious and takes food well. Extremely weak. Died April 16, 1872, suddenly.

(a) Cases in which convulsions were entirely or principally unilateral, and followed by hemiplegia of the same side :—

CASE 8.—A. H. (F.), æt. 41, admitted November 22, 1873, in an advanced stage of the disease. January 10, 1874.—Ten days ago had a seizure followed by coma. Patient had rigidity, then twitching, and finally paralysis of the left arm, which still continues. Has never recovered consciousness, and now lies in a state of deep insensibility. Pupils insensible to light; right is the larger. Died January 12, 1874.

CASE 9.—J. McC. (M.), æt. 37, admitted July 3, 1871. *General condition:* Is in the second stage. January 9, 1873.—Patient, who had been behaving much as usual yesterday, and had fed and undressed himself, was found at 9.30 P.M. yesterday to be in a semi-conscious condition, and to have twitchings of the muscles of the right side of the face, and to be partially paralysed on that side. This morning there is loss of power in the extremities of the right side; the face is drawn over to the left, and patient remains in a semi-conscious state. January 15, 1873.—Has slowly emerged from the 'status paralyticus.' On January 20, 1873, patient again became semi-conscious, the twitchings returned, and death occurred.

CASE 10.—J. A. (M.), æt. 38, admitted May 28, 1869. A marked case of the second stage of general paralysis. September 15, 1869.—Last night

patient had a paralytic seizure affecting the right side. This morning he is aphasic, and has spasmodic twitchings of the right side. September 22, 1869.—Has recovered from the local paralysis. Died April 23, 1872.

Case 11.—G. F. (M.), æt. 46, readmitted March 31, 1870. *General condition:* Advanced in second stage. January 13, 1871.—Had an attack of epileptiform convulsions yesterday, and was very sick. Vomited during the night. No better this morning; complains of pain in the head; is very stupid. Has been vomiting again. March 29, 1871.—At six o'clock this morning was noticed to be very stupid, and refused to get up. At seven o'clock A.M. he began to have spasmodic twitchings of the *left* side of the face, and slightly also of the muscles of the trunk and extremities of the *left* side. The left arm and leg are now paralysed. Just after midday the twitching ceased entirely, but patient continued very stupid. Temperature 99° in the evening on the sound side. March 30, 1871.—Pupils unequally dilated; left the larger; conjunctivæ injected. Slight twitching of the corner of the mouth. April 1, 1871.—Is now very restless; is continually falling through weakness. No return of the spasms. April 18, 1871.—To-day there is twitching of the *left* orbicularis palpebrarum, of the *left* corrugator supercilii, and of the *left* corner of the mouth. The limbs are also slightly affected in a similar way. The twitchings occur regularly, and number about 70 to the minute. Consciousness lost. April 22, 1871.—Has recovered consciousness, and is able to sit up again. April 26, 1871.—Has had a series of epileptiform attacks during the last two days. Is in a semi-comatose condition. *Left* side of the face is paralysed, and mouth is strongly drawn to the right. May 26, 1871.—This morning was again attacked with convulsions. Died at 2.45 A.M.

Case 12.—M. A. B. (F.), æt. 34, admitted September 30, 1872. *General condition:* In second stage of the disease. January 27, 1874.—Three days ago had a 'stroke,' followed by hemiplegia of the *left* side. February 6, 1874.—Has just passed through a series of epileptiform seizures affecting the *left* side of the face and the *left* extremities. Is now completely demented, and cannot understand a single word that is said to her. Died March 31, 1874.

Case 13.—J. L. (M.), æt. 36, a railway guard, admitted November 13, 1872. Stated to have received severe injuries in two collisions on the railway. Has been ill for ten months. *General condition:* Second stage of disease. December 15, 1872.—Has lost extravagant ideas; now very feeble; was yesterday only saved from choking by the timely removal of some pieces of gristle from the fauces. March 25, 1873.—Yesterday had two epileptiform attacks, resulting in loss of power of the *right* side. Is quite conscious this morning, but the loss of power continues on the *right* side. Pupils much contracted, especially the *right* one. May 7, 1873.—After last night, patient again became comatose, but rallied after some days, and gained sufficient strength to be able to get up daily and walk in the garden; was also able to recognise people, &c. Yesterday had a very severe and prolonged convulsive attack affecting the muscles of the whole body, but more especially those of the *right* side. This morning, however, is up again and going about as usual. Had an exactly similar attack a fortnight ago. June 28, 1873.—Yesterday had two severe fits, in which he fell and bruised his face; more weak and stupid to-day, but managed to get up.

July 8, 1873.—Had another seizure yesterday, and died at 11.30 A.M. to-day.

II. Cases in which both sides of the body were convulsed (either during the same attack or in different attacks), but not at the same time.

CASE 14.—H. W. (M.), æt. 38, readmitted July 27, 1871. *General condition:* Second stage of the disease. November 2, 1871.—Yesterday had an attack of depression. November 14, 1871.—Is extremely heavy and stupid; has ptosis of the *right* eyelid, flushing and comparative insensibility of the *right* side of the face; tongue protruded to the *right*. November 27, 1871. —To-day is observed to have twitchings of the *left* side of the body; is depressed, and can only with difficulty be got to take food. December 6, 1871.—Twitchings of the *right* side are again observed. December 26, 1871. —Is very thin and weak; mental condition very variable; has a well-marked purpuric eruption, which is confined to the lower extremities. January 4, 1872.—Eruption fading; patient is sinking. Died on January 7, 1872.

CASE 15.—M. L. (F.), æt. 35, readmitted October 5, 1870. *General condition:* Second stage. October 28, 1870.—Muscular movements very uncertain and jerky; tongue very tremulous; right pupil larger than the left. November 16, 1871.—Pupils much dilated; patient sits in one attitude continually, with a fixed stare. November 27, 1871.—During last night her *right* arm and leg became powerless; no drawing of the face; *right* pupil continues larger than the left; countenance very fatuous. August 21, 1872. —Yesterday had a severe convulsive attack, affecting the muscles of the *left* side of the body; there was marked increase of the temperature of the body, with a quick throbbing pulse; no difference in size of pupils; during the evening the twitchings of the muscles changed from the *left* to the *right* side for about an hour, and then returned to the left side: the *left* side since then has been continuously affected. April 26, 1873.—Both yesterday and to-day patient has been continually convulsed, first one side, then the other being affected; those seizures which have been observed have affected the *left* side chiefly, and during these the head has been drawn over the *left* shoulder; patient is only partially conscious. April 28, 1873.—There is no material change since last report; convulsions continue to recur from time to time, and patient frequently goes through gnawing movements of the jaw. April 29, 1873.—This morning was found to be going through gnawing movements again; head turned to the *left*, no material change; convulsions continue to recur at intervals. Died May 6, 1873.

CASE 16.—A. F. (F.), æt. 45, admitted February 14, 1872. Said to have been ill for about four weeks; is stated to have had a fit (how long ago not stated), and to have been low-spirited and rambling in conversation, though of quiet manners ever since. No family history of insanity, phthisis, epilepsy or apoplexy. Is much demented, and quite incapable of answering questions; she articulates slowly, as if the tongue and lips were not quite under control; she has none of the appearance of an epileptic; rather above average height; complexion dark; eyes brown; pupils unequal, the left being of about average size, and larger than the right, which is contracted; is rather thin; there is a distinct fine tremulous movement of the eyes; gait

slightly hesitating; heart, area of cardiac dulness somewhat less than the average; the heart sounds at both apex and base are accompanied by murmur, but in the latter situation there is decided softness and weakness of the systole; pulse quiet; tongue clean; liver and spleen, dulness normal. February 23, 1872.—Case of general paralysis in the second stage. April 2, 1872.—Had an epileptiform seizure with convulsions; left side of the body was most convulsed, face being turned over left shoulder; breathing stertorous; frothing at mouth; when seen was unconscious, the stertorous breathing having gone; she was lying in position of person after epileptic fit; she afterwards had six attacks similar to the first, but has not had another since this morning; she is now lying quietly in bed; makes no sign of recognition, but seems conscious, as she follows the movements of those about her with her eyes. April 3, 1872.—Patient seems a little more intelligent, otherwise is much the same as yesterday; pulse soft; no loss of power in any of the limbs; vomits frequently, and will scarcely swallow. April 15, 1872.—Becomes more and more demented every day; her speech very thick and heavy; has had no recurrence of the epileptiform seizures. May 11, 1872.—Has had another seizure; rigidity of one side well marked. May 21, 1872.—Yesterday at 5 P.M. patient had a convulsion affecting face and right arm, the left arm being stiff and rigid; in about an hour this was followed by a second convulsion, and again in an hour by a third; when seen twitchings of the left side of the face were observed, and strabismus of the right eye. During the intervals of the convulsions slight spasm of the facial muscles was observed; pupils unequal, the right being smaller than the left, but both contracted; pulse 135; temperature 102°; 10 P.M. temperature 99°; pulse 120. Has had several convulsions, and has continued in a state of semi-coma; at present is able to swallow, but can take little nourishment; pulse 125; temperature 101°. May 22, 1872. To-day very feeble; the twitchings of the face and strabismus continue; eyes bleared; conjunctivæ injected; face has a dusky hue; lips pale and the breathing more stertorous and oppressed from mucous accumulation in the larger bronchi; 6 P.M., little change is noticed in general appearance and condition; is now almost quite unconscious and shrinks but little on touching the conjunctivæ; pulse 136, small but fairly regular; temperature 103$\frac{2}{5}$°. May 23, 1872.—But little change has taken place; if anything, her face is more dusky and the twitchings less frequent; pulse 160; temperature 103·3°. Died 2.30 P.M.

Epileptiform Seizures affecting the right and left sides of the body alternately.

(Translated from 'Epileptiforme und apoplectiforme Anfälle der paralyt. Geisteskranken.' Westphal. Beobachtung I.)

'CASE of F . . . . e, æt. 38, admitted December 16, 1864. March 29, 1865.—'At 11.41 in the morning patient, who had previously let his head hang forward and was sleeping in his seat, suddenly fell forwards and was seized with general convulsions; immediately afterwards the following state was

noted :—Head turned towards right ; long-continuing twitch-
ings of facial muscles ; the eyes are rapidly and alternately
firmly closed and opened ; the orbicularis of the *right* side
appearing to partake the more strongly ; the mouth is drawn
to the right by the rapidly recurring twitchings ; clonic
twitchings in the arms and left leg ; the right leg is tetani-
cally rigid. Scarcely have these phenomena discontinued
when a new fit begins, in which the head is now turned to
the left; the left eye is firmly closed, and the mouth drawn
to the left. At the same time there follow general tetanic
spasms with general and pronounced cyanosis after the clonic
convulsions. The arms are in this case slightly flexed, the
hands closed, and the thumbs firmly clenched. In like manner
there follows a third fit, or rather a whole series of fits, suc-
ceeding one another and of definite type. At first the head
is drawn slowly towards the right; the mouth while under
convulsions is pulled strongly towards the same side, the right
eye strongly closed, the left slightly or not at all; after about a
quarter of a minute the head is turned slowly to the left, the
tonic spasm of the right orbicularis is relaxed by convulsions
affecting it alone; the eye opens, while now the left eye is
firmly closed, and the mouth drawn towards the left. During
this, slight convulsions take place in the extremities, and
certainly stronger on that side towards which the head is
turned. Similar attacks succeed one another continually after
short intervals, and in such a manner that the fit always sets
in with drawing of the head towards the right side, and with
corresponding contraction. Countenance pale, occasionally
cyanotic, left pupil somewhat wider than the right, both of
average size. 11.55, temperature 102·84° ; the spasms con-
tinued in the same manner with similar changes until 12.35
p.m.; immediately after patient lies quietly as if asleep, eyes
half closed, pupils contracted. 12.35, temperature 104·18° ;
2·40, has remained insensible ; skin moist ; temperature
102·56°–3.45, temperature 101·66°–6.45, temperature 101·12° ;
still remains as if asleep ; mouth drawn somewhat to the
right ; does not answer to a call, though he twitches when
pricked on the face or soles of the feet. March 30, morning,
still lies in a drowsy state ; does not respond when called, and

only slightly to a pinch; offers strong opposition to examination of his chest. March 31, looks up when spoken to; left eye opens last or sometimes remains closed; does not answer. April 1, answers questions. Seizure, August 2; suddenly fell from seat on which he had been sitting; half an hour with head hanging down as if asleep. On examination is found to be senseless and motionless, not to be roused by calling; placed in bed he lies with legs flexed, head somewhat backward to right, strong trembling of upper extremities and fibrillary twitchings of the pectoral muscles. The tremblings recur spasmodically, the lower extremities not taking part; the raised arms do not fall as if flaccid, but only slowly; to the attempt to bend them only slight though appreciable opposition is made; eyes to the right; pupils of average size, not acting; no response to pricking. About 4.30 P.M., October 6, a convulsive attack observed, the right half of the face and both right extremities were involved in tonic spasm, which gave way to clonic convulsions of the affected muscles; eyes directed upwards to the right. When patient was seen above an hour later, four or five similar seizures had taken place, according to the attendant. The following appearances present themselves: he lies upon the right side, eyes fixed upwards to right and wide open. Suddenly the right eye begins to close slowly (as described above), presently all the facial muscles of the right side fall into tonic spasms, the mouth is drawn high upwards and to the right, the right corner of the mouth appearing to be opened. Then a strong contraction of the right sterno-mastoideus occurs and drawing of the head towards the right, and directly marked extension and slight abduction of the right arm, fist spasmodically clenched, and lastly, the joints of the thigh and knee of the right side are spasmodically flexed.'

FUNCTIONS OF THE THALAMI OPTICI.

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THE opinion long entertained upon somewhat insufficient grounds, chiefly anatomical, that the optic thalami are the great foci of the sensory nerves, and that their grey matter constitutes the common sensorium or centre for sensations, has received of late powerful corroboration. The carefully-conducted experiments of Dr. Ferrier have established the fact that destruction of one of these ganglia is followed by complete anæsthesia of the opposite side of the body; and the less perfect and precise, but still instructive experiments of disease, are, I think, slowly revealing to us the same truth. Nothnagel has indeed asserted, as the result of experiments upon rabbits, the details of which have not yet been described, that the extirpation of these organs causes no direct disturbance of the cutaneous sensibility. But he adds that they have nothing whatever to do with the innervation of voluntary movements, and inclines to the belief that they are in intimate relation with the muscular sense.

Dr. Bastian, also, in his recently-published lectures on Paralysis from Brain Disease, has summarily rejected the hypothesis which ascribes sensory functions to these ganglionic masses, and has expressed his belief that it is discredited by pathological facts, and by the researches of Dr. Broadbent. But it is a noteworthy circumstance that Dr. Bastian, while denying to the optic thalami their alleged function as sense centres, has not suggested for them any other rôle in the

performances of the nervous system, and has not supplied any very definite rules to guide us in diagnosing a lesion limited to these ganglia. He has not referred to any cases in which, with a destruction of one optic thalamus, there has been loss or impairment of the sensibility of the skin on the opposite side of the body, but he admits that impairment of sensibility is perhaps slightly more marked in lesions of the optic thalamus than in those of the corpus striatum.

It is a question, however, whether Dr. Broadbent's researches, referred to by Dr. Bastian, really do discredit the hypothesis that ascribes the reception of the impressions of common sensibility to the optic thalami. By these researches it is shown that the crura cerebri consist of two layers of fibres, separated by the locus niger—a superficial layer called the crusta, and essentially a motor tract, and a deeper layer, called the tegmentum, and essentially a sensory tract. But it is further shown that these two layers, after passing the collar of the crus, expand in a fan-like manner, the crusta taking an anterior and the tegmentum a posterior position; the former, speaking generally, being in contact with the corpus striatum, and the latter with the optic thalamus. True, Dr. Broadbent points out that while fibres of both crusta and tegmentum can be proved to terminate in the corpus striatum, the diverging fibres of the tegmentum appear to pass onwards under the optic thalamus without ending in it. But he does not speak dogmatically upon the point, and is careful to add that it is possible that communications, by means of cell processes, do exist between the radiating fibres and the overlying ganglion, bringing them into a relation equivalent to the direct termination of fibres in cells. Dr. Broadbent also points out that the fibres of the optic thalamus, which radiate upwards and outwards towards the convolutions, accompany closely the diverging fibres of the tegmentum on which they rest, and with which they become intermingled. The complex arrangement of the fibres proceeding from the central ganglia to the hemispheres has as yet precluded the attainment of strictly accurate information as to their distribution, but Dr. Broadbent's able and elaborate investigations appear to indicate that the fibres from the

corpus triatum have a special area of expansion in the neigh-
bourhood of the posterior ends of the frontal gyri, and that
those from the optic thalami have special destinations in the
temporo-sphenoidal and occipital lobes. This is in striking
harmony with the results of Dr. Ferrier's experiments, and
with his conclusion that the whole brain is divided into a
sensory and motor region corresponding in their anatomical
relations with the optic thalami, and the corpora striata and
the sensory and motor tracts. It is also in harmony with
the interesting observations of Laborde. He maintains that
ramollissement of the surface of the brain seems to be nearly
always attended with a similar affection of one or other of the
cerebral ganglia, and he has satisfied himself that, in the
great majority of cases, the part of the cortex which is af-
ected will determine the site of the softening of the central
ganglia; thus, if the surface of the anterior left lobe be
softened, there will be also softening of the anterior part of
the left corpus striatum and if the left temporo-sphenoidal
lobe be softened, the left optic thalamus will participate in
the ramollissement.

But other anatomical indications of the nature of the
functional differentation of the great ganglia at the base of
the brain are not wanting. Both these great ganglia derive
their blood supply from all three of the main cerebral
arteries, but the relative proportions in which they are
served with blood by these arteries are very different. Thus
the corpus striatum derives its supply principally from the
middle cerebral artery, and receives only small twigs from the
anterior and from the choroid branch of the posterior cerebral
arteries; whereas the optic thalamus draws its chief supply
from the posterior cerebral, and only small auxiliary cur-
rents from the middle cerebral and anterior communicating
arteries. But the middle cerebral, the chief artery of the
corpus striatum, is distributed to the motor regions of the
cerebrum, while the posterior cerebral, the chief artery of
the optic thalamus, is distributed mostly to the sensory
regions of the cerebrum. These facts surely suggest the
intimate association of the corpus striatum with motor, and
of the optic thalamus with sensory functions.

And the revelations of diseases seem to me to confirm the suggestions of anatomy with reference to the functions of the cerebral ganglia. In a Lecture on Cerebral Hæmorrhage, published in March, 1874, I recorded an observation which I had then several times repeated that in cases of clot, involving the optic thalamus, there is much more uneasiness and restlessness during the period of unconsciousness than in other cases of clot in which the corpus striatum is alone implicated. I also reported what I had upon more than one occasion noticed, that after the recovery of consciousness in cases of apoplexy, pain in the head and in the limbs of the paralysed side is complained of often when the optic thalamus has been the seat of extravasation, and seldom when the corpus striatum has been its locality. These observations which, since the publication of the lecture alluded to, I have had opportunities of repeating, are, I am aware, at variance with received notions, which attribute cephalgia in cases of organic diseases of the brain to superficial, and not to deep causes. No doubt it is true, as a rule, that severe headache is more frequently connected with changes in the textures enveloping the cerebrum than with changes in the cerebrum itself; but still there are, I am convinced, instances in which lesions of the optic thalamus, not sufficiently large to occasion much pressure on the meninges, and unassociated with any perceptible morbid modifications of the meninges themselves, are accompanied by intense protracted and deep-seated cephalgia. Such instances at least create the suspicion that it may be a sensory centre, injury of which thus occasions pain; while those other cases in which a similar lesion is associated with pains in the paralysed limbs, independent of muscular spasm, corroborate that suspicion, and afford what is probably an illustration of erroneous nervous reference.

When describing my observations as to the existence of headache and peripheral pain in lesions of the optic thalami, I also referred to the destruction of sensibility which occurs on the side of the body opposite to such lesions. Türck, quoted by Carpenter, gives four cases of apoplexy or ramollissement of the brain, in which, with either transient or permanent hemiplegia, there was intense and permanent

anæsthesia of the same side of the body. The whole of the cerebrum was sound, except in all these cases the external part of one optic thalamus, where was a lesion of the cerebral substance of considerable length, though not in most instances extending before or behind the optic thalamus. My own observations quite accord with those of Türck; and indeed carry me further, leading me to conclude that in extensive injury of one optic thalamus it is not merely the sensibility, but the reflex excitability of the opposite side of the body that is abolished or held in abeyance. My experience has convinced me that whenever the substance of one optic thalamus is broken down or softened to any considerable extent, peripheral irritation fails to induce, not only signs of sensory stimulation, but even responsive movements in the limbs subtending that thalamus. By the light of that experience I have been able, in several cases of hemiplegia, to predict with accuracy the situation in which the clot would be found after death. Whenever ordinary paralysis of one side, of a pronounced character has associated with it annulment of reflex action and of sensibility in the powerless extremities, I do not hesitate to conclude that the central lesion is localised in or extends into the optic thalamus; and whenever paralysis of the same kind co-exists with unimpaired or but slightly enfeebled reflex activity and sensibility, I feel tolerably certain that the morbid change is confined to the region of the corpus striatum. The enfeeblement or abolition of reflex excitability in those cases in which the optic thalami are implicated may affect both the upper and lower limbs, but almost invariably it is found to exist in the feet and legs to a much more marked degree than in the hands and arms. Pinching and squeezing the toes, and applying heat, cold, and electricity to them, will sometimes fail to cause the slightest movement, while the same treatment of the fingers will be tardily followed by muscular contractions.

The frequency with which cerebral hæmorrhage involves several of the great ganglionic masses at the base of the encephalon, and with which successive hæmorrhages obliterate or confuse the records of early and definite lesions, makes it a comparatively rare occurrence to meet with a case in which

the distinctive effects of a clot in the optic thalamus can be traced out in a thoroughly satisfactory manner. Such a case, however, occurred in the West Riding Asylum quite recently, and as that case presented features of peculiar interest I shall quote it at length :—

Hannah B——, aged 50, a widow and housewife, chargeable to Leeds, was admitted to the West Riding Asylum, on October 27, 1874, and was then labouring under slight dementia, characterised by forgetfulness, confusion of thought, and consequent trepidation. She had been a temperate woman, and had a good family history, and her individual experiences offered no explanation of her mental ailment. She had, however, just passed through the change of life. Up till March, 1875, she underwent no material change, continuing somewhat stupid and perverse, but on the 31st of that month she suffered a slight apoplectic seizure, accompanied by drawing of the mouth to the right side, temporary loss of speech, and weakness of the whole of the right side of the body. There was no loss of consciousness, and within a few hours of the seizure power had fully returned to the arm and leg, although there was still some impairment of speech, with slight inclination of the tongue to the right when protruded. From this attack she recovered satisfactorily and remained well, complaining only occasionally of headache, until July 9, when she had another seizure very similar to the previous one. Consciousness was not lost, and the enfeeblement of power was confined to the right leg, in which general sensibility was decidedly deadened. From this second attack she also rallied, so thoroughly that no permanent weakness could be detected in the side which had been paralysed. On the morning of August 23 she was conversing in the visiting-room with some relations, her reunion with whom had occasioned her some joyous agitation, when suddenly her speech became thick and her statements unintelligible. In four minutes she dropped from her seat, quite unconscious, and with complete hemiplegia of the left side. Being conveyed to bed, and having had croton oil administered, she improved somewhat in the course of a few hours, and was able to recognise and name in a somewhat thick spluttering voice those who were waiting upon her, but towards evening she again became deeply comatose, and remained in that state, breathing heavily but not stertorously throughout the night. On the morning of the 24th she was still unconscious, and it was then noticed that the face was drawn to the right, and reflex excitability was altogether lost in the left foot and leg. When the foot was tickled, pinched, and pricked, it remained motionless, but when the same stimuli were applied to the right foot it was at once moved and withdrawn. When the right hand was pinched similar phenomena were noticed, but when the left hand was pricked no disturbance was occasioned. When the right cheek was pricked there was a suppressed groan, when the left cheek was pricked there was no visible result. Deglutition could not be performed, so that nutriment had to be given by enemeta. During the two following days improvement took place. The power of swallowing returned, and some degree of consciousness, so that she could be roused up, and made efforts to

speak, but the left leg and arm remained powerless and insensible to impressions of any kind. On the evening of the 26th some fresh hæmorrhage probably occurred, as she agaiu became deeply comatose, and breathed stertorously. There was slight external strabismus of the left eyeball, but no ptosis. Both pupils were strongly contracted, and the left cheek was pendulous. The pulse was 116, aud the temperature on the paralysed side 103°, and on the sound side 102·6°. A warm bottle of moderate temperature which had been applied to the feet, wrapped in flannel, had produced some pemphigus-looking blisters upon the left foot, but had in no visible manner affected the right one. On the 27th she was much in the same state, except that there was marked nystagmus of both eyeballs. At 2.30 in the afternoon of that day she died rather suddenly. At the post mortem examination, held twenty-six hours after death, there were found thin films of blood extravasated iuto the meshes of the pia mater, over the tips of both tempore-sphenoidal lobes and over both lateral lobes of the cerebellum. The large arteries at the base were remarkably atheromatous, being dilated and in some places surrounded by patches of an opaque dirty yellow colour. There was slight cloudiness of the arachnoid over the frontal aud parietal lobes, but no thickening of the pia mater, which stripped readily from the convolutions. The surface of the brain was somewhat flattened. Ou slicing down into its substance, a large dark red clot mixed with shreds of broken-down nervous tissue was found, occupying the place of the right optic thalamus. It projected into the lateral ventricle, and filling up the third ventricle, touched the optic thalamus of the left side. Externally it was bounded by the tœnia semicircularis and corpus striatum, the substance of which remaiued intact, and posteriorly it occupied the descending coruu of the ventricle. On removal, it was found to rest on the fibres of the crus cerebri of the right side. The whole of the right optic thalamus had been broken up by the clot, which had not, however, extended its ravages into surrounding parts. Iu the left lateral ventricle there was a little fluid blood, and in the fourth veutricle there was a small clot, which was continued upwards through the iter into the third ventricle. In the outer part of the optic thalamus of the left side there were two small united clot cavities, together about the size of a horse-bean, and containing detritus of a blackish brown colour.

In this case the limitation of the clot to the right optic thalamus was singularly distinct. The whole bulk of that ganglion was broken down, while surrounding parts were scarcely interfered with, and the symptoms therefore observed during life may be received as characteristic of a destroying lesion of that part of the encephalon. These symptoms included, first, incomplete coma; second, paralysis of the limbs on the opposite side of the body, face, and tongue; third, some impairment of articulation; fourth, loss of general sensibility on the opposite side of the body and face; fifth, abolition of reflex excitability on the opposite side of

the body. Now, of these symptoms it is the last two that seem to me to be distinctive of a lesion in the optic thalamus, and that are absent when the corpus striatum alone is involved. The following case illustrates the symptoms which are exhibited when a destroying lesion is confined to the latter ganglion :—

William B——, aged 67, widower, a labourer, chargeable to Bradford, was admitted to the West Riding Asylum, on October 28, 1872. His history set forth that he had been a keen politician for many years, and had undergone much excitement in attending Reform and Chartist meetings, as well as in indulging in drink, to which he had been long addicted. He was suspected to have suffered from delirium tremens more than once. Twelvemonths previous to his consignment to the Asylum, he became restless and unmanageable, wandering about at night, and threatening to kill the woman with whom he was living, and he was then taken to the Bradford Workhouse, where he continued to be quarrelsome, noisy, and intractable. He was at times incoherent in his conversation. No hereditary taint was known to have existed in his family, and he had never suffered from injury to the head, epilepsy, or paralysis. When admitted he was much excited, talking incessantly, and incapable of giving rational answers to questions, but readily moved to tears. He said he was 22 years of age. He was in good bodily condition, and his circulatory, respiratory, digestive, and genito-urinary systems were found to be normal. The pupils of the eyes, however, were unequal, the right being the larger of the two. Two days after admission he had what was believed to be a threatening of a paralytic stroke, becoming suddenly pale, husky in his voice, and unsteady in his gait. From this condition, however, he speedily recovered, and remained tolerably well until April 17, 1873, when a distinct stroke occurred. There was stupidity, aphasia, complete loss of power on the left side without impairment of sensibility, and marked drawing of the mouth to the left. The stupidity gradually deepened into coma, and on the following day, April 18, he was quite unconscious and breathing stertorously. The pupils were much contracted. When the left arm was raised it fell heavily, like a dead weight, but when the right arm was raised it fell less precipitately and heavily. Reflex excitability was maintained unimpaired. Both legs moved about instantly and violently when the soles of the feet were tickled, and both hands were quickly withdrawn from pinching or any irritation. The pulse was 126 and compressible. On April 19 and 20 the same observations were repeated, and on April 21 the patient died. At the post mortem examination, forty-three hours after death, the dura mater was found to be strongly adherent to the frontal bone, while the large arteries at the base were enlarged in calibre and atheromatous to a very remarkable degree. There was cloudiness and opacity of the arachnoid over the frontal and parietal lobes, the convolutions of which were somewhat wasted, and there was a softened patch in the anterior half of the right corpus striatum. Round this patch, in which the cerebral substance was reduced to a mere fluid pulp, there was a zone of slight vascular engorgement. In no other part of the brain was

any softening clot or other structural change discovered, but everywhere the minute vessels were exceedingly coarse. The inner surface of the aorta was rough and atheromatous, and both lungs were œdematous. In the right kidney were three small cysts.

Here, as in a case to be afterwards alluded to, there was serious disruption of the corpus striatum, sufficiently large to cause motor paralysis, but with that disruption there was no curtailment of sensibility, nor interference with reflex excitability, such as was witnessed in the case previously recounted, where the optic thalamus, as well as the corpus striatum, was broken down. Here also there was emotional incontinence, a proneness to be easily betrayed into tears or laughter—a condition which I have more frequently found associated with lesions of the corpus striatum than with lesions of the optic thalamus.

Occasionally the different effects of lesions of the optic thalami and corpus striata may be seen in one and the same case, and these may be so clearly defined as to justify a prediction as to the situation and extent of the changes in these ganglia. In the following case I was able to say during the life of the patient that, in all probability, the right optic thalamus was exempt from those destructive changes which had evidently involved the left optic thalami and both corpora striata :—

John I——, aged 55, single, a dyer, from Dewsbury, was admitted to the West Riding Asylum on July 25, 1874. He was reported to have had a stroke a month prior to admission, and to have been deranged in mind ever since. He had been intemperate in his habits. When examined he stated that about a year ago he had had a stroke, after which he was unconscious for a day. Previous to and after the stroke he felt a numb sensation in the right temple and right side of the nose and upper lip. This had continued to annoy him more or less ever since. He understood all that was said to him and replied promptly, and with reference to remote events his memory seemed to be good ; but about the order, duration, and dates of recent occurrences he became much confused. He thought he had been in the Asylum for five days when he had resided in it only twenty-four hours, and he could not name the day or month. He was very emotional, and burst into tears whenever his mother's death was referred to. During the night he had hallucinations of the senses. He was in fair bodily condition, and his pupils were equal. He was unsteady on his legs and swayed from side to side, especially when first starting to walk. The general sensibility of the skin was unimpaired, but he still complained of disagreeable sensations

in the right temple and side of the face, and also of watering of the right eye. The heart's impulse was very feeble. On August 6 it was noted that he was becoming more demented. He could not tell where he was; he complained more than ever of pain in the right temple and cheek; his mouth was slightly drawn to the right, and he dragged both legs in walking, but the left most so. There was impairment of grasping power in both hands. His speech was slow, and his articulation indistinct. He said he had often a sense of deadness and prickly feelings in the fingers of both hands. On August 8 he was worse and could not stand. Reflex action was normal in the left foot and leg, but defective on the right side. On August 26 he was feebler in body and mind; he could not speak nor move about, and had a fatuous expression of countenance. On September 13, at 6 P.M., he had in the presence of a medical officer a convulsive fit of great severity, the first recorded in his case. His head and eyes were turned to the left; there was rigid extension of the arms and some rigidity of the legs, which were in a semiflexed position. After the rigidity came clonic spasms, affecting face and limbs, and most marked in the right arm; there was also a gnashing movement of the lower jaw. There were a succession of fits after that first one. Each fit lasted about fifteen seconds, and there were scarcely any perceptible intervals between them. The incursion of the fit was generally accompanied by a faint cry. It was the right side that was affected. Every third or fourth fit, the left arm became rigid; at other times it lay flaccid. In the intervals between the fits the head was drawn towards the left shoulder, but as soon as the fit commenced it was turned to the right. The fits continued to recur at the rate of two a minute until 11 P.M. Then the bowels, having been previously cleared out, an injection of chloral was given, after which the fits were reduced in frequency. Till 1.35 they came at the rate of one a minute, and after that, till 2.35, at the rate of one every five minutes. At that hour a second chloral injection was administered, with the effect of again widening the inter-convulsive intervals. The fits recurred somewhat irregularly, but about one in every ten minutes until 4.20, when there was an entire cessation of them. The head was then drawn to the left, the pupils were much contracted, and there were occasional uneasy and twitching movements on the right side. Reflex excitability was abolished in the right foot and arm, but was present on the left side. Deglutition was performed without difficulty; the pulse was 78 and of fair strength, and the respirations were 30. On the morning of the 15th he had twelve more fits like those above described, and on the evening of that day he died. The post mortem examination was performed forty-seven hours after death, and showed that the skull was considerably thickened, and that the arachnoid was milky over the frontal and parietal lobes. The pia mater was tough, but stripped freely, and the basal arteries presented many large patches and rings of atheroma of a dirty whitish-yellow colour. The intra-ventricular pear-shaped portion of the left corpus striatum had disappeared, and its place was a tough fibrous membrane. The greater part of the substance of the left corpus striatum and optic thalamus was broken-down, pulpy, and of a pale fawn colour, and at the outer border of these bodies in the medullary substance of the hemisphere there was an extensive area of softening. The intra-ventricular portion of the right corpus striatum was broken

down, pulpy and discoloured, but the extra-ventricular portion of this ganglion was sound as well as the right optic thalamus. The walls of the left ventricle of the heart were thickened, and the cardiac valves were atheromatous, while the inner surface of the aorta was rough, eroded, wrinkled, and contained some calcareous deposits, besides much atheroma.

Sometimes the symptoms described as indicative of a destroying lesion of the optic thalamus are developed in such a manner as to reveal with tolerable distinctness the progress of the disintegration of that ganglion. In the following case it seems to me that the extravasation of blood began in the corpus striatum near the hand and arm centre, and that the optic thalamus became implicated when pain in the head was first felt. The disappearance of reflex excitability from the left leg probably marked the complete destruction of the optic thalamus. I do not recollect to have witnessed convulsive seizures in any case of cerebral hæmorrhage in which the clot was strictly confined to the optic thalamus, nor have I encountered those early tonic and clonic spasms in the paralysed limbs, and about the face and neck, which have been alleged to be especially frequent in lesions of that ganglion. It will be remembered that experimental irritation of the optic thalami, which has been many times practised by numerous observers, has always failed to induce convulsions, and that in the rotatory movements which have followed section of one optic thalamus, the animal has always remained standing, and has not fallen, as has been almost invariably the case when similar movements have been brought on by injuries of other parts of the brain.

William John S——, aged 34, married; attendant in the West Riding Asylum, was a sober, steady man, who had never had any serious illness. During 1873 he had a distinct attack of gout affecting the great toe of the left foot, and in the beginning of 1874 a similar attack occurred. For several months towards the close of 1874 he complained of frequent pains in the head, dull in character, and not limited to any particular region, but affecting now the forehead, now one side, and again the back of the head. He suffered also from several attacks which were supposed to be bilious in character, and which were characterised by intense headache, and long-continued retching and vomiting of bilious matter. Latterly he had been somewhat pale, but had not lost flesh, and had been able to go about his work cheerfully and energetically. On Wednesday the 25th November, 1874, he performed his duties as usual, but complained to his wife of altogether unwonted mental depression, which passed off, however, in the evening. At

9 P.M., with his wife, he accompanied a friend who had called into Wakefield, walking a distance of about three-quarters of a mile. The weather was intensely cold, but he was warmly clothed, having on a great-coat, and remarked that he felt quite warm. When he reached Wakefield he went into his friend's house, and there sat chatting in a warm room for about three-quarters of an hour. He partook of a few cockles, but had nothing to drink. At five minutes before ten o'clock he rose to return home, put on his great-coat, and was lighting his pipe, when he suddenly turned to his wife and said, ' My left hand feels quite dead.' He began to rub it with his right hand, and then said he had lost the use in his left arm. He became deadly pale, and turned to sit down, and as he did so his left leg slipped from under him and became powerless. He asked that a doctor might be sent for, and it was noticed that his speech was thick. He then seemed to be conscious and intelligent, and told his wife in a thick, husky voice, that he was certain he had had a paralytic seizure. Dr. Wade was sent for, and when he arrived the patient was still partially conscious. He complained of frightful pain in the right side of his head, which had then just come on, vomited some cockles, and then had a convulsive attack. Several other convulsive attacks occurred in rapid succession. After each he recovered consciousness, and complained of numbness and loss of power in the left arm and leg ; his complaints, however, became gradually less strenuous, and it was noticed that he grew drowsy and lethargic. At 10.40 he was placed in a cab and sent to the Asylum. When lifted out of the cab, at 10.55, he was deeply comatose, his face was pale, but not at all drawn to either side ; the pupil of the right eye was much dilated, and that of the left eye was much contracted. His breathing was stertorous and at the rate of forty respirations in a minute. His pulse was 78, somewhat irregular, but full and incompressible. The left side of the body was perceptibly colder than the right side. There was distinct response to stimuli in the right arm and leg, and a very slight response in the left leg, but none in the left arm. The bladder had emptied itself involuntarily. All the muscles were flaccid, and there were no clonic spasms, but there were from time to time restless, uneasy movements of the arms consentaneously upwards and downwards. Sinapisms were at once applied to the legs and a hypodermic injection of ergotin was administered, but no mitigation of the symptoms took place. At 11.15 there was no response whatever to stimuli on the left side. The breathing became more laboured, the pulse more irregular, the jaw dropped, and saliva flowed over the lips. At a quarter before twelve o'clock he was turned on to his side, when enormous quantities of frothy and blood-tinged fluid flowed from the mouth and nostrils. The respirations were now fifteen per minute, with distinct intervals. The eyeballs had become strikingly prominent, and were indeed standing out of the head, so that they could not be covered by the eyelids. At ten minutes to twelve the left pupil suddenly dilated. The breathing then became gasping in character, and the pulse slower. The last breath was drawn at about five minutes to twelve and the heart went on beating for forty seconds longer, when it suddenly stopped also.

Post mortem examination, twenty-one hours after death :—The body is

exceedingly well nourished; there is a thick layer of fat over the abdomen; no external marks, bruises, nor injuries are visible. Rigor mortis is present in a marked degree, and there is a large amount of hypostatic congestion. The skull is of average thickness and density, but is not quite symmetrical; it bulges a little posteriorly on the left side. On removing the skull the meningeal vessels are seen to be considerably engorged. When the dura mater is reflected, the brain is seen to present a peculiar flattened appearance. All the gyri are flattened, and the sulci are obliterated, as if the brain had been subjected to distension. In the sulcus, immediately behind the posterio-parietal lobule on the right side, there is beneath the arachnoid, in the meshes of the pia mater, a patch of dark-coloured blood. The flattening and smoothing of the brain above referred to is most marked on the right side, although distinctly visible in the left hemisphere also. The upper surface of the brain is very pale and anæmic, as if the vessels of the pia mater had had all the blood squeezed out of them by the distended brain. The whole brain (uncut) weighs 1565 grammes. The brain has a dry appearance externally. There is a line of attachment between the two hemispheres along the gyrus fornicatus, and here the brain substance is arched upwards (as well as the corpus callosum) by some pressure from beneath. There is no atheroma of the large vessels at the base. The arachnoid is not at all thickened, and the pia mater strips freely. On slicing down the right hemisphere, a large dark clot of blood is found breaking up the brain substance to the outside of the right lateral ventricle. A great portion of the right corpus striatum, and the whole of the optic thalamus and an immense tract of the medullary substance of the right hemisphere, is broken down, so that a huge irregular cavity is formed, from which 55 grammes of dark clot are removed. The cavity and clot are extra-ventricular and extend into the white matter of the frontal lobe, and posteriorly break into the descending cornu of the ventricle. The clot comes nearest to the surface in the middle part of the parietal lobe, where it actually trenches upon the grey matter. There is a good deal of sanguineous fluid in the ventricles. The brain substance, where not implicated in the clot, is singularly pale and anæmic. Pericardium empty. On the outer surface of the heart, over the ventricles, there are several white glistening patches, slightly raised, of irregular shape. The heart weighs 515 grammes; the walls of the left ventricle are enormously thickened and enlarged. The valves are competent. The lungs are quite healthy. The right kidney weighs 83 grammes. The left 99 grammes. The capsules of both are everywhere intimately adherent, tearing away the renal substances when removed and leaving a rough granular surface beneath. The kidneys have externally a puckered appearance. The cortical substance is much wasted; in the right one, it has almost disappeared. At the upper part of the right there are a few small cysts.

The three following cases illustrate clearly the grouping of symptoms which accompany serious organic mischief in the optic thalamus alone and associated with other lesion :—

Mary B——, æt. 53, who had been an inmate of the West Riding Asylum since 1858, and who was labouring under delusional insanity, was seized soon after going to bed on the night of September 15, 1873, with violent and deep-seated pain in the head. She became pale, faint, unconscious, and lost power entirely in the right arm and leg. On the morning of the 16th she was conscious, but confused and sluggish in all mental processes, while there was slight impairment of muscular power in the right arm. The face was not drawn to one side, but the tongue when protruded had a tendency to the left; the pupils were slow in their movements, and the right was somewhat larger than the left, but not dilated. There was a want of reflex action on tickling or pricking the feet on both sides, but most marked on the right, and distinct defect in the localisation of impressions. Vomiting occurred at intervals, and the patient sighed and yawned frequently. After aperient injections, sinapisms to the stomach, and a diet of milk and lime-water, improvement was inaugurated, and on September 26 it was noticed that the vomiting had ceased, that power had almost been regained in the arm, but that severe pain in the head was still complained of. On October 2 the report was that the headache still occasioned distress, and on November 7 the entry was to the same effect. On January 22, 1874, it was recorded that she had improved, but that power was still deficient in the right arm and leg, and that a sense of numbness and deadness, with pain, was constantly felt in these parts, as well as in the head. On February 7, Mary B—— died after the occurrence of another cerebral hæmorrhage in the pons Varolii, and on post mortem examination the source of the symptoms above described was discovered in a clot cavity in the left optic thalamus. This cavity, of the size of a horse-bean, and containing a pulp of very dark-brown colour, lay in the line of the left choroid plexus, and burrowed into the substance of the optic thalamus, which immediately around it was somewhat hardened.

David R——, aged 44, married, a cloth-finisher, chargeable to Leeds, was admitted to the West Riding Asylum on February 25, 1874. He had always enjoyed excellent health until 1869, when he failed in business, a circumstance that weighed greatly upon his mind. Nothing, however, occurred to indicate failing health until the beginning of 1871, when one day his face was seen to be drawn to the right side, while his speech was at the same time thick. For a few weeks the right side of his body was slightly affected, but this gradually wore off. His memory was distinctly weakened, and he was occasionally childish. About nine months later, at the close of 1871, he had a more severe attack of paralysis on the same side, and this was followed, fifteen months later, in the spring of 1873, by another stroke. The last stroke prior to his reception into the Asylum occurred in October, 1873. After each stroke his mind became manifestly weaker, and during 1873 he became possessed by various delusions, chiefly concerning money matters. His father died of apoplexy, and his father's brother was insane. When interrogated on his admission he answered readily, but rambled in his statements, and was childish in his manner. He was aware that his mind was not what it used to be, that his memory failed him, and that trifling matters caused him disproportionate annoyance. He confessed that his mind was often occupied by strange undefined fancies. His voice

was thick, indistinct, and of peculiar tone. Reflex action was apparently impaired in both legs, most so in the right. Cutaneous sensibility was normal, muscular power in the two hands and arms was equal, but there was dragging of the right leg. The face was not drawn to either side. A month after his admission he was found in bed in the morning in an insensible condition, out of which he could not be roused. His right arm was rigidly flexed, and his right leg was rigidly extended. Tickling the soles of the feet, applying heat and cold, produced no reflex movements in the right arm and leg, but were followed by immediate movements on the left side. The eyelids were tightly closed, and the pupils equally dilated and inactive. The pulse was 130 and very weak. On March 24 he was slightly better. He was partly conscious, but did not speak. He used his left arm freely, and moved his right one (now flaccid) occasionally. He swallowed well, and partook of plenty of nourishment. On March 25 he was restless. The right eye remained constantly open, and the left one was closed. The right pupil was much larger than the left one. The right arm was flaccid but powerless, and was not moved away from irritation, such as promptly caused withdrawal of the left arm. The mouth was drawn to the left side. On March 28 there was complete coma, with marked drawing of the face to the left, powerlessness and insensibility of the left side of the face and of the left arm and leg. Death took place on March 29, at 2.45 P.M. The post mortem examination was made forty hours after death. The whole of the convolutions of the cerebrum had a compressed and flattened appearance, most marked on the left side. On the under surface of the cerebellum was a thin film of fluid blood. When the left hemisphere was sliced down, a large dark recent clot of the consistence of currant jelly was displayed, occupying the position of the corpus striatum and optic thalamus, the substance of which was entirely broken down. The clot projected into the left lateral ventricle. In the right ventricle was some red fluid, but no clot. The septum lucidum remained entire, and a plug of clot filled the foramen of Monro. The basal arteries were thickened, but not atheromatous. There was slight hypertrophy of the left ventricle of the heart, and the kidneys were both contracted and granular, and had adherent capsules.

Jane G——, 49 years of age, a housewife, was admitted from Leeds on June 13, 1873. The history of her illness was, that sixteen months previous to her admission she had suffered a stroke, which deprived her of consciousness for a fortnight, and of power in the right side of the body permanently. After that stroke it was found that she had altogether lost the use of her right arm and hand, that her right leg was much crippled, and that her articulation was greatly impaired. It was also found that her temper, at all times an irritable one, had become worse, and that her mental faculties were generally enfeebled. Subsequent to the first stroke, at intervals which have not been ascertained, two other strokes occurred. One of these caused temporary complete loss of speech, and the other paralytic weakness of the left side, while both of them seemed to hurry on mental deterioration. Her memory became utterly untrustworthy. Her affections were deadened, and delusions and hallucinations haunted her. She thought that a policeman was concealed under her bed, and that her room was full of strange people. When brought to the Asylum she was found to be

incapable of answering any but the simplest questions, and to these she replied in monosyllables. Her past life was a blank to her. She was altogether confused in her ideas, and had an obvious difficulty in remembering words as well as in articulating them. She was quiet and orderly however, wore a cheerful expression, and seemed to enjoy life in a vegetative fashion. Physically she was below the average height, had grey hair and eyes, and was stout and well-nourished. On her cheeks large dilated vessels ramified, and in her eyes irregularity of the pupils was apparent, the left visual orifice being very much larger than the right one. The right arm was quite powerless, and was kept permanently and rigidly in a flexed position, and the right leg was also powerless and rigid. Reflex excitability in these parts was diminished and sensibility was decidedly blunted. The fingers and toes might be pricked or pinched without any expression of pain ensuing. In the left arm and leg there seemed to be a certain degree of loss of power. When in repose, the two sides of the face were quite symmetrical, but speaking or laughing disturbed their equilibrium. Whenever the muscles were thrown into action the mouth was drawn over to the left side. The urine was albuminous. Throughout the summer J. G—— grew gradually worse. Reflex excitability vanished altogether from the right leg, and was diminished in the left one. The tongue when protruded pointed to the left, and the lines of expression were obliterated on the right side of the face. She sank and died on November 29, 1873. At the post mortem examination, thirty-nine hours after death, the whole of the left optic thalamus was found to be broken down, and indeed a large irregular cavity with pulpy contents occupied the site of that ganglion. Along its outer side, and also along the outer side of the left corpus striatum, was a broad line of softening of the medullary substance of the cerebrum, with four small distinct clot cavities, with brownish pulpy contents. In the intraventricular part of the left corpus striatum there was another long irregular clot cavity. When the right hemisphere was sliced down, three or four minute clot cavities with yellowish contents, the largest not bigger than a pin's head, were revealed on the outer border of the right corpus striatum and optic thalamus. There was thickening of the walls of the left ventricle of the heart, and contraction of the kidneys.

These cases, it seems to me, would of themselves give a very high degree of probability to the hypothesis, that the optic thalami are sensory ganglia, and have the same relation to the nerves of common sensation as the olfactory, optic, and auditory ganglia have to their respective nerves. They are of importance, as showing that the facts of disease are in accord with the revelations of experimental inquiry, and they are of value as affording hints and suggestions which may be of service in further investigations into brain function.

It will be noticed that in one of the cases described in this paper, in which there was destruction of the optic

thalamus, there was slight elevation, while in another case there was decided lowering, of temperature on the paralysed side.   In the case in which the elevation was noted the observation was not taken until three days after the formation of the first clot, when perhaps some fringe of inflammatory reaction surrounded it; but in the case in which the lowering was noted, the observation was made immediately after the formation of a large clot ploughing up both the great ganglia. In that latter case the fall of temperature on the paralysed side was very striking.   The left arm, leg, and side of the body felt quite chill, while the right arm, leg, and side were of normal warmth, if not above it.   The transition too from the chilliness of the left to the warmth of the right side was very abrupt in the middle line of the abdomen and thorax. It seemed not unlikely that in this case there was intense irritation of some vaso-motor centre, causing anæmia and deficiency of animal heat in the dependent parts of the body. Uspensky (Virchow's Archives, 1866) has indeed localised such a centre in the immediate neighbourhood of the optic thalamus.   He has reported four cases in which hemiplegia was associated with alterations in temperature of the palsied parts.   In the first case there was palsy of motion and less complete palsy of sensation in the right half of the body, and a curious reflex excitability, smart percussion of the skin of the right or of the left side (anywhere except in the head or flanks), producing convulsive actions of the muscles of the right side.   The temperature of the palsied parts was on an average 1·3° C. below that of the other side, and sometimes the difference was as much as 4° C.   In a second case there was complete hemiplegia, loss of motion and sensation, impairment of intelligence, contraction of the pupil on the palsied side, and an entire absence of all reflex excitability of the paralysed muscles.   The temperature of the paralysed side was constantly from $\frac{1}{2}$° to 1° C. higher than that of the other side.   The case terminated fatally in three months, and after death softening of the brain substance external to the left lateral ventricle was discovered.   The conclusion which Uspensky draws from his observations and experiments is, that there are vaso-motor centres in the brain,

probably in the situation of the softening in the case just
adverted to.   Irritation of this centre would cause anæmia
and a fall of temperature on the opposite of the body, and
paralysis of it, hyperæmia and a rise of temperature.   Fleish-
man has recorded a case of cerebral tubercle in a boy two
years old, commencing in the left thalamus opticus and ex-
tending into the crus, in which both these conditions were
seen alternately. The symptoms were exactly those described
by Afanasieff in section of one cerebral crus.   They were
partial paralysis of the left oculo-motor nerve, dilatation of
the pupil, which was unaffected by light, paralysis of some of
the facial muscles, and of the trunk and extremities of the
right side ; tremor of the right hand, increased cutaneous
sensibility of the right side with the exception of the head,
ophthalmia, hyperæmia of the retina, choroid, and optic disc
of the left eye, incapacity of retaining stools or urine, and
abnormal fluctuations in temperature.

It is interesting to note in this connection that in hysteria,
modifications of cutaneous temperature are very often asso-
ciated with modifications of sensibility.   A rapid fall of tem-
perature, with paleness of the surface, may accompany
anæsthesia and analgesia in that disorder, and may be fol-
lowed by as rapid a rise, accompanying redness of the
surface, subjective feelings of warmth, and copious perspira-
tion.   When contemplating phenomena of this kind in hyste-
rical cases in which there were also mental perversity, loss of
consciousness, and even convulsions, it has occurred to me to
speculate whether hysteria might not depend upon an unstable
condition of the nerve-elements in the region of the optic
thalamus, just as chorea probably depends upon instability
in the region of the corpus striatum.   However this may be,
the fact remains that in hysteria, especially of the hemiplegic
type, singular fluctuations of temperature are associated with
hyperæsthesia and anæsthesia, and point to intimate relations
between the sensory and vaso-motor centres.   And there
are other facts which point in the same direction, such as the
occurrence of pemphigus, or rather perhaps of pompholyx,
in cases in which there is abolition of sensibility of one half
of the body.   The sudden appearance of large transparent

bullæ upon one of the limbs betokens some grave interference with the cutaneous circulation of the part affected, and the association of such a condition with loss of cutaneous sensibility owing to a centric lesion, at least gives feasibility to the hypothesis that the vaso-motor and sensory centres are in juxtaposition.   There was distinct pompholyx of the paralysed foot of Hannah B., and a considerable number of similar cases have fallen under my notice, some of them quite lately.   Sarah H., who has for several years been completely paralysed on her left side, but in whom reflex activity was little if at all impaired, presented a few days before her death a crop of blebs upon her left hand and arm.   In her brain were found three small clot cavities, with gelatinous contents in each corpus striatum, and a belt of softening to the outer side of the right corpus striatum, extending outwards and backwards into the medullary substance of the hemisphere, but not encroaching upon the optic thalamus.

The first case of this kind which I saw made a deep impression on me, as it nearly led me into making what would have been an unjust accusation against an attendant.   A hemiplegic patient was shown to me with numerous large transparent vesications over the paralysed foot, and these so much resembled the effects of a scald that I fancied that there had been carelessness in bathing him, and that his foot had been placed in excessively hot water, until similar large blisters formed on the leg almost under my own observation, and until I noted more exactly the small amount of febrile disturbance that accompanied the local condition, and the small amount of redness which surrounded the cuticular elevations.   In that case, as in most others of a like kind that I have encountered, there was disintegration in and around the corpus striatum and optic thalamus.   Indeed, I have never seen acute pemphigus or pompholyx, or whatever this condition may be designated, except in hemiplegic patients or in general paralytics.

Unfortunately, in all the cases which have been quoted in this paper the state of the pupils was not recorded, but in three of them the tendency was towards dilatation of the visual aperture, most marked in the eye of the same side

as the injured thalamus. This corresponds generally with the results of experiment which have shown that sight and the action of the pupil may be retained even when both optic thalami have been removed, and that these ganglia may be irritated without any contraction of the iris ensuing. The nystagmus which occurred in the case of Hannah B. had a special interest in connection with the small superficial clots which were found on the lateral lobe of the cerebellum. I have never seen any impairment in the co-ordination of the muscles of the eyeball in disease confined to the optic thalamus.

But these cases which have been quoted, and the observations for which they afforded opportunities, have an interest altogether beyond their pathological significance, and indicate the necessity which exists for a review of our received notions respecting reflex or diastaltic action. They contravene a general law, hitherto implicitly accepted, that reflex centres manifest increased activity whenever the influence of higher regulative centres is cut off from them. They show that the reflex centres in the spinal cord are even less independent than they have been supposed to be, and that relations of an unexplained character subsist between them and the cerebral ganglia.

No doubt can be entertained that both in man and animals reflex actions are most freely and characteristically displayed when the spinal cord is entirely disconnected from the cerebrum. A transverse section of it by the knife or by disease imparts a vivacity, never otherwise seen, to the irritational movements of these parts, whose afferent and efferent nerves are derived from it below the point of section. Whoever has witnessed the violent movements of a decapitated frog, when its feet are touched or when the cloaca is tickled, must have been impressed by the much greater excitability of the spinal centres under such circumstances, than when the cerebrum remains connected with them. Whoever has observed a case of paraplegia, dependent upon degeneration or injury above the lower dorsal region, must have been struck by the exaggerated, and sometimes almost convulsive character, of the reaction set up by the application of mode-

rate stimulation to the lower extremities when these are totally deprived of voluntary power and of sensibility. It has been customary to explain this heightened reflex activity in segments of the cord isolated from the cerebrum in one of two ways. In the first place, it has been supposed that the nerve force liberated by the received impressions under such circumstances is discharged in its whole volume by the associated afferent nerves, no portion of it being withdrawn for transmission to the encephalon, as must always be the case when the integrity of the cord is preserved. And, in the second place, it has been supposed that when the cord is cut across the liberation of nerve energy in it on the reception of an appropriate impression is unrestrained by a controlling influence sent forth from the encephalon, which permeates it whilst it remains entire, and that the discharge of energy is therefore more widespread and decisive.

Reasoning by analogy, we should have inferred that the destruction of the optic thalamus, which appears to be equivalent, in so far as the cord is concerned, to a section severing one half of it from communication with the highest centres, would be followed by an increase of reflex excitability at least in the half thus cut off from cerebral intercourse. The destruction of a thalamus implies an interruption in the conveyance of sensory and motor currents between the superior and inferior centres, and places the latter therefore in a position in which all the energy awakened by impressions may be directly expended in movement, unweakened by a cerebral subsidy, and in which no voluntary restrictions can neutralise the free play of its inherent functional activity. When a thalamus has been placed *ab agendo*, we should certainly expect the reflex excitability of the opposite half of the cord to be perceptibly increased, at any rate until secondary changes in its structure had been established. But the very reverse of this is what we invariably find. The reflex excitability of the cord, instead of being increased is diminished, instead of being brought up to a pitch of high intensity it is altogether abolished. Sensory impressions cannot pass upwards, for the hemiplegic limbs are destitute of feeling, but no augmented transverse

reaction is witnessed. Voluntary prohibitions cannot pass
downwards, for by no effort of will can the palsied limbs be
moved, and yet none of the license of unconstrained activity
is exhibited. Ideational, emotional, sensori-motor move-
ments are impossible, and yet excito-motor movements are
not exuberant. By numerous observations it has been made
certain that the destruction of the optic thalamus is followed
by abolition of reflex activity in the limbs of the opposite
side of the body, and that partial disintegration of an optic
thalamus is followed by impairment of the reflex activity of
the limbs of the opposite side of the body.

In speculating as to the way in which lesions of the
optic thalami interfere with reflex activity, and as yet
nothing but speculation is possible, the first suggestion that
presents itself is that the optic thalami may be not merely
sensory, but inhibitory centres ; and that the irritation set
up by laceration and rupture of their substance may be suffi-
cient to hold in check effectually the activity of subser-
vient centres. Every segment of the cord with its pair of
trunk-nerves is joined by longitudinal commissural fibres,
with segments above and below, and by centripetal and
centrifugal fibres with relatively higher centres. Of the
latter, the centrifugal fibres, many travel from the optic
thalamus probably to every segment of the cord, and certainly
to those important and highly independent segments which
receive the massive nerves of the limbs. Along these centri-
fugal fibres it might well be that inhibitory as well as motor
influences are transmitted. Indeed, it seems certain that the
optic thalami, if not themselves inhibitory centres, are at least
traversed by, or are in immediate contact with, some nerves
having that function that are connected with still higher
centres, and are distributed to lower ones which they regu-
late and control. Might it not be that irritation of these
nerves arrested the libero-motor function of these lower
centres ; and might this not be so, even supposing that
these nerves were broken off from their ganglionic connec-
tions ? The matter forming the axis-cylinder of a nerve is
not restricted to the mere transmission of molecular motion,
but has the power of giving out or intensifying it, and so

shares to some extent the function of the vesicular portion
of the nervous system.   When an efferent nerve is irritated
near its muscular termination the effect is slight, but when
it is irritated at a point more remote from its periphery a
more decided result is obtained.   And just as the length of
the nerve through which the irritation has to pass is in-
creased, so is the magnitude of the effect augmented, from
which it is clear that during the activity of a nerve there is a
multiplication of the molecular motions received and trans-
ferred by it.   This multiplication or accumulation of nerve
force in the act of transmission is due to the isomeric trans-
formation of a disturbed nerve, and the liberation of motion
which concomitantly takes place.   Now the distance between
the optic thalami and the reflex limb centres is considerable,
and it might well be that a comparatively slight irritation of
the centrifugal nerves proceeding from or through the great
sensory ganglia to lower reflex centres, even when they are
broken off from their proper libero-motor apparatus, might
be so intensified in transmission as to place these centres in
a state of complete disability.   And it is a noteworthy fact
that the impairment of reflex excitability is always most
marked in the lower limbs, the reflex centre for which is more
removed from the optic thalami, and is therefore more likely
to be powerfully affected by any nervous irritation that
is intensified in transmission.   And the irritation necessary
to set in motion the gradually accumulating inhibitory
influence might well be derived from the mechanical injury
done by a clot, and the rearrangement of particles which
that must imply, from secondary inflammatory changes
around the extravasated mass, or from the instability of
nerve matter which pressure within the cranium must
always occasion.

No sooner, however, has this hypothesis of inhibitory
influence transmitted from the broken-up optic thalamus to
the reflex centres presented itself, than serious objections to
it also start up.   It is a destroying and not a discharging
lesion with which we have to deal in cases of clot in these
bodies, and the effect of a destroying lesion on an inhibitory
centre must be the abolition and not the intensification of

inhibition. If the optic thalami were inhibitory centres, the result of extensive inroads on their substance would inevitably be greater freedom and not greater limitation of reflex action. Then the effect of a destroying lesion upon inhibitory nerves merely passing through the optic thalamu would also be in the direction of locally diminished restraint and locally increased reflex activity; but the moment that a clot is formed in the optic thalamus, the deterioration in reflex action is apparent. That is not postponed until irritation is set up, but is recognisable instantly. Then if the mere rupture of inhibitory nerves could interrupt reflex action in the cord we should probably have the same result following upon extravasation into the corpora striata, which are probably traversed to much greater extent than the optic thalami by these nerves; but this is not the case, for in apoplexy of the corpora striata the reflex excitability is not interfered with, and if mere rupture of efferent nerves could in any way maintain them in a state of constant functional activity we should have permanent cramp and spasm of the limbs whenever the motor tracts in the corpora striata were severed by extravasation, but neither is that the case. In some instances, as in the case of Hannah B., the entire mass of the optic thalamus has been broken up and converted into pulp, and yet reflex excitability has been abolished, which could scarcely have been so had the optic thalamus been an inhibitory centre or an inhibitory highway.

Another theory explanatory of the impairment of reflex excitability in lesions of the optic thalami which suggests itself, is that there is an intermediate inhibitory centre placed between the spinal cord and the optic thalamus, which any serious injury of the latter throws into a state of higher activity. It may be that every higher centre is inhibitory of that centre which is immediately below it, and that the sensori-motor ganglia are regulative of the centres of compound co-ordination—the medulla oblongata, pons varolii, and corpora guadrigemma—which in turn exercise a check upon the centres of simpler co-ordination in the spinal cord ; and if this is so, it is clear that the destruction or incapacity of the optic thalami would enable the enlarged and differential

continuations of the spinal cord within the cranium which have been named, so powerfully to control the spinal centres as virtually to abrogate their functions; and feasibility is given to this theory by the fact that in lesions or disorders of those centres of compound co-ordination—the corpora guadrigemma, pons varolii, and medulla oblongata—such as might be supposed to weaken their powers, the reflex activity of the spinal cord is generally greatly increased, just as if it were freed from all inhibitory restraint. Then, again, it is an established fact that the cerebrum, the next higher centre above the sensorial ganglia, maintains a potent control over them, and that it is only when the activity of the cerebrum is suspended that the full reflex operations of these ganglia can be displayed. Without, however, assuming what analogically seems not improbable, the existence of a definite progression in inhibition corresponding with the complication and differentation of other nervous functions, it is still possible to believe that it is an intermediate inhibitory centre that is responsible for the results seen in the sphere of reflex action when the optic thalami are damaged. Supposing such a centre to be situated between the cord and the optic thalamus, although with no functional dependence upon the latter, it is easy to conceive that it might be made to discharge itself inordinately, and so to quench all reflex action in the cord by pressure exerted upon it by the swollen or distended ganglion. Pressure of such a degree as to induce anæmia in nervous tissue without mechanical disturbance, will cause excessive discharges of energy, at least until innutrition begins to tell; and so pressure, such as may result from clots in the optic thalamus, brought to bear upon an inhibitory centre in the pons varolii or medulla oblongata, might for a time at least cause the passage of nerve force from that centre with undue facility. Plausibility is lent to this view by the observation that the impairment of reflex action in lesions of the optic thalami is always in proportion to the extent of the lesion; that is to say, to the size of the clot by which it is invaded. When the clot is large and occasions considerable pressure, reflex action is generally extinguished; when it is small, and occasions less pressure,

reflex action is only diminished. It is to be borne in mind, however, that this observation may have other meanings attached to it, and that the amount of injury done to the optic thalami themselves, and not the pressure exerted upon adjacent bodies, may be the measure of reflex impairment in such cases.

But various grave difficulties discountenance the acceptance of this theory of an intermediate inhibitory centre hyper-active, when lesions in the optic thalami interfere with reflex spinal action. It is well known that nerve-tissue is not capable of continuous stimulation or continuous discharge, and it is not therefore likely that any inhibitory centre could act presistently in such a manner as to keep the reflex centres inactive for prolonged periods, as is the case when the optic thalamus is reduced to ruin. We should expect to have intermittent inhibitory control, but this is not so, for at no moment in cases such as I have described, where the whole thalamus is involved, can any reflex excitability be demonstrated. There are never any stray reactions such as we might look for in a sound centre strongly inhibited ; there are no indications of relaxation of inhibition, no movement is ever elicited in spite of inhibition, or while it is momentarily slackened. The phenomena point to some failure in the reflex process itself, rather than to any excess of repression exercised over it.

And still another interpretation of the facts connected with the abolition of spinal reflex action, with destruction of the optic thalamus, may be found in the supposition that impressions which have reached the cord do not habitually react at once upon the motor machinery, but are first transmitted upwards to higher centres, whence they are bent backwards upon the muscular apparatus. Allowing to the spinal cord a power of independent action, directly and commissurally, it may still be that it generally acts reflexly through or in association with a superior centre. The sensorial ganglia can undoubtedly act alone in a reflex manner, but they almost invariably consult the cerebrum before dealing with the impressions which they receive ; and so it may be that the spinal cord, though capable of spontaneous reaction, may yet

commonly refer to some higher seat of compound co-ordination before sending forth au answer to any message brought to it; and, if this is so, a very imperative law of diffusion of nerve energy would be speedily set up. The shock of molecular disturbance received in the unstable grey matter of the cord, when an afferent nerve is changed by some application to its outer end, would diffuse itself mainly in the channels of habitual distribution aud would be propagated, not at once through clusters of corpuscles to efferent nerves, but first upwards through uore involved communications; and then having turned downwards and outwards to the spinal foci and thence to the instruments of expression, and this course or current of nervous diffusion having been fully established, would, under certain circumstauces, be interrupted with difficulty. Of course, if the cord were by any means cut across, the diffusion upwards would be summarily prevented, and the whcle of the euergy liberated by a stimulus, conveyed by an afferent nerve, would be at ouce trausferred to an efferent one, and would be so carried off to the muscles, but if the cord remained intact and an interruption in continuity existed in the encephalon, then the ceutripetal fibres of the cord would convey upwards as usual impressious derived from the periphery in solicitation of aid or guidance from the encephalic centres, and that energy would be dissipated and wasted at the very point where it ought to have been reflected or bent backwards. No return of energy would be obtained by the spinal cord, and no muscular movement would be possible, whether it was iu the disintegrated optic thalamus itself, or in some intermediate centre thrown out of gear by its disintegration that the breach of continuity took place, the result would be pretty much the same. A stimulus to the surface would induce little or no reflex movement.

Reference has been already made to the physiological truth, that in all ordinary reflex acts there is the passage of a certain amount of the nerve force disengaged in the grey matter of the spinal column to higher reservoirs of molecular motion. By this fact, as we have seen, is often explaiued that increase of automatic activity which is witnessed when the spinal column is severed from higher ganglionic centres, and

when no passage upwards being permitted, all the nerve
force generated in the cord is expended in reflex actions.
But this current upwards, resulting in higher reflex co-
ordinations in sensation or in ideation, after the stimulation
of a spinal centre, has been supposed to be simultaneous with
the current outwards which calls into play reflex muscular
contraction. No suggestion has been offered as to the mode
in which the amount of energy liberated is divided, so that
one portion of it is despatched upwards and another portion
outwards ; nor has it been made intelligible how the relative
proportions of the two different currents are determined.
The existence of the two currents, however, is certain, and their
simultaneity has been assumed. But if under normal con-
ditions the two currents should not be simultaneous but
successive, if the nerve disturbance transmitted to the higher
centre were first despatched, and that sent to the muscles
were only sent forth when a message of liberation was re-
ceived from above ; if, in fact, what may be termed the
encephalic loop current were an integral part of every ordi-
nary reflex act, then the influence of an intra-cranial lesion
in checking reflex action would not be difficult to under-
stand. It would be no objection to this view that there
would be a sacrifice of time in the transmission of the loop
current, for as nerve force travels at the rate of at least
twenty-eight yards per second, the reaction to a peripheral
stimulus would still be practically instantaneous. The
rapidity of the whole process, in the transmission of nerve
disturbance and the manifestation of its effects, is such that
its appreciation of the different steps involved in the process
is scarcely possible. In experimenting upon myself, how-
ever, I have sometimes thought that when the toe is pricked
the sensation of pain actually precedes the movement of
withdrawal, and in experimenting upon patients with
sluggish nervous systems, I have certainly noticed upon
many occasions that after pricking of the toe the little cry of
pain has anticipated the muscular contractions of the leg.
Now this cry of pain is a secondary reflex act, through the
sensorial centre ; it is the result of a discharge from efferent
nerves from the summit of what we have spoken of as the

encephalic loop-line in reflex action, and we should not certainly expect that it would be developed earlier than the primary reflection upon the motor apparatus, unless indeed what we have regarded as the primary reflection really itself took place by way of the loop-line. However this may be, it remains indubitable that in every reflex action, while the spinal cord remains entire, the disengaged molecular motion is not wholly and solely discharged along efferent nerves, but that at least a portion of it is propagated upwards to centres of a superior grade when further changes are inaugurated, which are in turn still more widely diffused. Although we may not as yet be justified in dogmatising as to the paths of diffusion which these further changes ensue, there are, I think, valid reasons for believing that one of these paths conducts downwards to the reflex centres of the cord. The whole nervous system is pervaded by reflected and re-reflected disturbances, and it seems to me at least probable that certain waves of nervous change are incessantly flowing downwards from sensorial to reflex centres, and are essential to the full functional activity of the latter. The same waves are probably instrumental in maintaining muscular tonicity, for when the sensorial centres are destroyed the muscles of the opposite limbs appear to be altogether thrown out of action to be absolutely paralysed. While a patient in whom the optic thalamus has been broken up by a clot lies deeply comatose and paralysed, the powerlessness of the limbs of the two sides will be found to be very different. If the arm opposite the lesion be raised, it will drop utterly helpless and flaccid ; but if the arm on the same side as the lesion be raised it too will drop, but by no means so flaccidly as the other. A certain amount of firmness and elasticity remain in the one which have departed from the other, and this might indeed be made a basis of diagnosis as to the side of the cerebral lesion, in cases in which no other information was procurable. The fact indicates, I think, that undulations of nerve force are being perpetually diffused centrifugally from or through the optic thalami, and that being so, no great effort is required to conceive that these undulations are a necessary part or condition of reflex

excitability, and that their withdrawal means the abolition of that excitability, and their enfeeblement its deterioration.

But whatever explanation we may adopt of the co-incident abolition or enfeeblement of sensibility, and reflex activity in the paralysed limbs in destroying lesions of the optic thalami, the fact is an important one, in its physiological as well as in its pathological relations.

THERAPEUTIC VALUE OF CHLORAL HYDRATE IN EPILEPTIC CONVULSIONS.

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An apology seems almost needed for any remarks on the subject of chloral, about which so much has been written, but, although the drug has been extensively used in convulsive disorders, outside of lunatic asylum practice, it does not seem to have been exhibited in such forms of convulsive diseases as are incidental to lunatic asylum patients in anything like the degree commensurate with its merits. The writer's experience of it, first at the Durham County Asylum, and subsequently at the West Riding Asylum, extending over a period of several years, is so favourable as to induce him to bring it under the notice of his fellow-workers; hence this short paper. Almost immediately after its introduction by Liebreich, in 1869, chloral achieved an extraordinary notoriety, and was no doubt sometimes injudiciously and imprudently used both by the medical profession and the laity. The journals teemed with instances of its almost miraculous virtues, and it was hailed by the more enthusiastic of its admirers as a panacea for all the ills that flesh is heir to. As Dr. Crichton Browne wrote about it in the ' Lancet ' of April 1, 1871 :—' A river of chloral has flowed through the land, and all diseases have been indiscriminately immersed in it.'

This undue enthusiasm was naturally enough followed by a reactionary movement on the part of the more judicious

and cautious of the medical profession, and cases were not
wanting to prove that the new panacea, like most other
potent drugs, though capable of much good, yet at the same
time proved itself a very dangerous and treacherous weapon
in the hands of those who used it, without proper precaution
and advice.   This reaction was, however, carried too far, and
the caution induced thereby was perhaps excessive, though,
on the whole, the movement tended rather more to the im-
partial observation of the remedy, than did the extravagant
advocacy which brought the drug into disrepute.   It is
nevertheless very certain that chloral hydrate is still occa-
sionally abused, and cases not infrequently come to light
in which fatal results have followed its habitual use, espe-
cially where to the practice is superadded the inordinate
use of alcoholic stimulants.   In asylum practice the em-
ployment of chloral passed through the same phases as
characterised its introduction elsewhere; the early enthu-
siasm received the same check, and caution and moderation
succeeded to extravagance and indiscrimination.   This result
was probably sooner attained in Asylum than in general
practice, owing to a variety of causes, and chloral took its
proper place in the armamentarium of the alienist phy-
sician, a place of which it had proved itself worthy, and
from which it will not readily be ousted.

At first it was thought to be an absolute preventive of
excitement, and was administered in all such cases; but it
was soon found that the daily use of chloral to control the
excitement of chronic maniacs was surely followed by grave
deterioration of the physical condition of those patients.
Again, it was in those cases that the sudden deaths occurred,
which have been held out as warnings to the incautious, and
which have deterred some from using the drug at all; finally,
permanent good was rarely effected, since when the drug
was withheld the excitement returned as fiercely as ever.   On
the other hand, the excitement of mania transitoria, of mania
e potû, and the short outbursts of ungovernable fury which
frequently precede, accompany, or succeed to a batch of
epileptic seizures, all these are admirably met by a single full
dose of hydrate of chloral, and the improvement in the

two former classes of cases, at any rate, is decisive and often permanent. As for the alarming symptoms which have been observed to follow the exhibition of a single moderate dose, the writer cannot but think that they must have been unintentionally exaggerated, and the vomiting so frequently found by the earlier observers has never yet been seen by him after a close and constant observation of the action of chloral extending over a period of six years. Nor has it been noticed in such of the lower animals as the writer has seen experimented upon. Chloral having been so lavishly used in ordinary practice, it necessarily followed that convulsive disorders had a share of its favours, and here it was that the drug found its speciality, so to speak ; and the visible superiority of its action over all other forms of treatment, led to its general use in chorea, hooping-cough, puerpural and uræmic convulsions, and the convulsions of hydrophobia and tetanus. Amongst the earliest recorders of successful cases were Baron Paul de Leydewitz, of Basle ; Mr. Warren Tay, of the London Hospital ; and Mr. E. R. Denton, of Leicester.

Until the autumn of 1869 cases of rapidly recurring epileptic seizures, and the peculiar epileptiform attacks of some forms of organic brain disease, were treated in the Durham County Asylum [1] first by clearing out the bowels thoroughly by enemata, and then by giving large doses of bromide of potassium, or inhalations of chloroform. Occasionally, when the pulse was very full and rapid at the onset, repeated subcutaneous injections of green hellebore were practised with a remarkable influence on the tension and frequency of the pulse, but absolutely without effect as regarded the convulsions. The effect of the large doses of bromide of potassium was simply *nil*.

Chloroform inhalations were more satisfactory no doubt in controlling the number and severity of the convulsions, but their effect was very transient, ceasing as soon as the inhalation was discontinued, and necessitating prolonged administration of chloroform with its accompanying dangers.

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[1] The same treatment was probably used in most other Asylums, excepting the hellebore injections.

In September, 1869, Langenbeck successfully treated a case of tetanus, coming on after a lacerated wound of the face, with hydrate of chloral. Dr. B. W. Richardson stated in his lectures about this time that chloral promised to be useful in cases where increment of heat, *muscular spasm*, and pain were to be combated, and that it was worthy of an extensive trial in tetanus. At or about this time the writer met with several cases of repeated epileptic seizures, which proved rapidly fatal in spite of treatment, and he then resolved to try chloral hydrate in the very next case which presented itself. A general paralytic, in the last stage of the disease, was seized with epileptic convulsions during the night of the 24th of October, 1869; fit followed fit with great rapidity, and the convulsions were very general and severe. The patient became comatose, his breathing was embarrassed and his pulse uncountable; while his body perspired profusely. The bowels having been cleared out by enemata, ʒss of chloral hydrate was injected, and within a few minutes the convulsions ceased; the pulse and respiration fell rapidly, and the patient dropped into a deep sleep, from which he awoke free from convulsions. The effect of the drug in this case was almost magical, even exceeding the most sanguine anticipations, and several cases having been treated in a similar manner, and with the very same result, the writer became convinced that in chloral he possessed a remedy with which he might effectually control convulsions. His subsequent experience has confirmed this belief. A series of cases will now be given, illustrative of the action of chloral, and taken from the Case-Books of the Durham County and West Riding Asylums.

CASE 1.—E. D——, æt. 44, was admitted June 16, 1869. Powerfully built, well nourished, but inclined to corpulency, a general paralytic. Prevailing mental condition on admission, melancholic with suicidal tendencies. This condition was soon replaced by some excitement. Multiform delusions of an optimistic character were developed—*e.g.* patient is making millions of money, having secured the contract to supply Heaven with flour. His wife is the woman in the moon. He is filled with millions of grubs and worms, &c. October 11, not looking well for past day or two; stupid and drowsy; perspiring profusely without any cause. At 10 P.M. seized with first epileptic convulsion. Seen almost immediately afterwards by

reporter. Found unconscious. Pupils largely and equally dilated. Breathing rapid and semi-stertorous. Skin hot, but moist. Pulse 100, full. Bowels had been moved. No paralysis of either side. At 10.15, second seizure. Convulsed as follows : first eyes opened, rolled from left to right, then fixed. Left arm raised, brought round to right side ; fixed in tonic contraction ; respiratory muscles also rigid. The clonic spasms engaged gradually all the muscles of both trunk and limbs, and the facial muscles ; patient churning bloody froth and mucus with the mouth and lips ; finally the convulsions ceased, leaving him snoring loudly and profoundly unconscious. At 10.30 P.M. the third seizure. At 10.40, the fourth. At 11.5, the fifth. At 11.20, the sixth. At 11.35, the seventh. At 11.59, the eighth. All these fits were about equal in severity and duration, and in them the spasms followed the same order. At 11.59 he seemed likely to become asphyxiated. Bronchial tubes gorged with bloody mucus and froth ; occasional feeble attempts at getting rid of it. Breathing interrupted. Heart's action laboured ; pulse not to be counted. Patient had an injection of thirty grains of chloral at about 10.30. No effect seemed to have been produced up to 11.59, when a second dose of thirty grains was injected, and the anus plugged, as part of the first dose had been ejected. At 12.15 the ninth seizure occurred. At 12.25, the tenth. Both these fits were equally severe with the former. At 12.35, the eleventh. In this, the clonic spasms were confined to the left upper and lower extremity and the right leg the arm of that side being free. At 12.50, the twelfth fit. In this the right half of the body was unaffected, save the muscles of the eyeball and face. At 1.25, the thirteenth fit, still less severe than the last, and patient passed into deep sleep, and did not awake until the afternoon of the next day, when he complained of giddiness, headache, and soreness of the limbs.

Patient had a return of epilepsy at 7 A.M. on August 1, 1871, but only two seizures occurred, and he survived until December 1, when he died asthenic.

Case 2.—J. S——, æt. 30, was admitted on October 2, 1869. He is of arthritic diathesis ; well nourished and muscular. Mental condition : partially demented, with frequent attacks of religious exaltation, accompanied by excitement and restlessness. A confirmed epileptic, epilepsy the result of cranial injury. Seizures rare, and preceded by vertigo for a day or two. February 4, 1871, had a rapid succession of fits to-day, which seemed likely to terminate in the status, and congestion of the lungs. Had ʒss dose of chloral by the mouth, and as the convulsions continued, and patient was unable to swallow, two injections were practised at half-hour intervals of eighteen grains each. An hour after he was sound asleep, with a full, slow pulse ; convulsions ceased.

Case 3.—W. G——, æt. 44, was admitted on June 1, 1870. Physical condition : well nourished and muscular. Mental condition : melancholic and demented. Addicted to masturbation for some months past, according to statement of his wife. December 8, more demented ; very ravenous feeder ; suspected to be a general paralytic. February 18, 1871, undoubtedly a general paralytic, motor impairment, and defective articulation very marked. Is becoming dirty in his habits. April 6, was found to be stupid this morning, and was allowed to remain in bed. At 7.0, had an epileptic

seizure. Was very severely and generally convulsed, and had wet the bed. When seen by reporter was found unconscious, but was moaning and tossing about. Bowels had been freely moved the day before. Pupils very much contracted and very sluggish. Patient had four more seizures before reporter was sent for again. Second, at 9.0. Third, at 9.25. Fourth, at 9.45. Fifth, at 10.5. He was visited at 10.10. Was found lying on his back, breathing rapidly and with effort; perspiring profusely, and had wet the bed again in the last fit. Sixth attack, at 10·30; very severe. First eyes rolled from right to extreme left, and remained fixed; next head turned from right to left; muscles of trunk and limbs became rigid in tonic spasm, which lasted a considerable time, patient's face becoming much discoloured; clonic spasms commencing in arms, and spreading gradually over the body, and continuing for an unusually long period. At 10.45 ʒss of chloral hydrate was given by the rectum, but only part of it was retained, and ten grains of chloral were given by the mouth with great difficulty at 11.5. The seventh fit occurred at 11.15 A.M.; not so severe. The eighth seizure occurred at 11.35. The ninth, at 11.52. Ten grains of chloral by rectum at 12.0. The tenth seizure at 12.15; not so severe. 12.30, ten grains of chloral by rectum. The eleventh seizure occurred at the same time, still less severe. The next fit at 1.3 P.M., not so severe as last. At 2·15 had still occasional seizures, much less severe, and only involving the muscles of the eyeballs and face. Twenty grains of chloral were given by the mouth, and a few minutes after patient fell into a deep slumber, and did not awaken for several hours, and had no return of convulsions. The congestion of the lungs consequent on the fit passed into pneumonia of the bases of both lungs, and went on to the formation of an abscess in the right base, the contents of which exhaled a most horrible fœtor, and necessitated the isolation of this patient from the other inmates of the sick ward. He became convalescent, and the motor impairment lessened considerably, and he was discharged at the request of his friends. He remained quiet and manageable until June, 1873, when he became excited and restless, and was certified for in order to be readmitted into the Asylum, but the epileptiform seizures recurred, and he died at home in the status epilepticus.

CASE 4.—H. F——, æt. about 50, admitted March 28, 1870. An organic dement with frequent excitement. Bodily condition: well nourished; considerable arterial degeneration. September 4, suffered an attack of hemiplegia; is drowsy, but not unconscious. September 15, still in bed, and had several severe epileptiform seizures which went on after a first dose of ʒss of chloral, but ceased on the administration of a second, patient remaining sound asleep for several hours.

CASE 5.—J. G——, æt. 43, admitted on March 26, 1871. Bodily condition: is a general paralytic, fat and muscular. Mentally, much excited; multiform delusions of the usual optimistic character June 10, motor impairment increas ingrapidly; much more demented. September 28, has had several epileptic seizures following each other rapidly, the clonic spasms never passing off until the tonic spasm of the next fit commenced. Most of the muscles of the trunk and limbs seem engaged. The bowels were cleared out by enemata, and thirty grains of chloral given per anum, the rectum being plugged in order that the drug might be retained altogether. Con-

vulsions ceased shortly after September 30, clonic spasms confined to face and arms, returned on the 30th, and patient died exhausted.

CASE 6.—R. B——, æt. 36, admitted August 6, 1873. Well-marked general paralytic, just discharged from the Glasgow Royal Asylum. Bodily condition: well nourished, and of average muscularity; considerable motor impairment. Mental condition, considerably demented. On August 22 he had a slight amount of hemiplegia, which soon passed off. On the 25th had a short outburst of excitement, with great restlessness and much shouting. September 15, sent to the Infirmary, failing rapidly. October 8, a severe epileptic seizure at 7 P.M., followed by clonic twitchings of left limbs. Second fit at 7.5, and another every five minutes until 8.0, when reporter saw him. There is no cessation of the clonic spasms, which are merged into the tonic spasm of each seizure. Left limbs alone engaged. Bowels having been thoroughly cleared out, ʒss of chloral was given by rectum and the anal orifice plugged with lint.

Patient at this time was comatose, breathing heavily. Moist, loud râles audible all over chest, and even the larger tubes are filled with frothy mucus. 11.30 P.M., he has had four seizures since administration of chloral. To have another ʒss dose. Only two seizures followed the second administration, and they occurred at longer intervals, after which patient slept for several hours. October 10, there was a return of the fits, which yielded again as before. October 17, patient died of dry gangrene of the left lower limb; no pulsation could be detected below the femoral.

The following brief extracts from the reports of a series of cases are taken from the West Riding Asylum Case-Books:—

CASE 1.—G. L——, æt. 20, a confirmed epileptic, admitted October 29, 1872. January 13, 1875, an explosion of fits occurred, and was followed by constant muscular agitation and jerking about of the limbs. Thirty grains of chloral were given; movements ceased.

CASE 2.—B. R——, æt. 18, admitted September 21, 1874; has been almost in status epilepticus for two days past. Last night had eight fits from 7 to 10 P.M.; fifteen grains of chloral produced sleep almost immediately. No return of fits. May 2, 1875, has had twelve seizures since bedtime yesterday evening; 6 A.M. given forty grains of chloral; went to sleep almost immediately afterwards, and had not another seizure.

CASE 3.—H. E——, æt. 47, admitted June 13, 1873. A general paralytic. May 26, 1874—At 8.15 this morning seized with epileptiform convulsions. These were confined to head, face, and left upper extremity. Head rolled to left, eyeballs oscillating; clonic spasms without remission until 8.45. when ʒss of chloral dissolved in warm water was injected into the rectum. In five minutes the convulsive movements of left arm diminished and gradually ceased. The movements of the face went on for three minutes longer, then diminished, and finally ceased, the occipito-frontalis and orbicularis palpebrarum being the last muscles to become quiescent. The left arm remained powerless for sometime.

CASE 4.—H. D——, æt. 15, admitted September 8, 1873. A confirmed epileptic. August 4, 1874—A sudden and furious outburst of excitement culminated at 4.15 P.M. in a very severe fit, which was rapidly followed by others.

At 4.45 he had had ten fits in all, and the seizures succeeded each other after intervals of half a minute. Breathing much embarrassed by bronchial secretion. Forty grains of chloral given by œsophageal tube. For the next fifteen minutes fits occurred as frequently as before, but were less severe. At 5 P.M. twenty grains more were given in the same manner. Three seizures followed at greater intervals; finally at 5.12 he fell asleep, and the seizures did not recur. These convulsions were very severe and followed this order: First eyeballs oscillated, turned to right; next orbicularis oris and palpebarum twitched; then head turned to right. Right arm next involved, then left in an equal degree, then right leg, finally left lower limb.

CASE 5.—J. B——, aged 42, admitted November 10, 1873. An epileptic of six weeks' standing. On the night of April 8 be had rapidly repeated seizures until 10 P.M., when he had ʒss of chloral; two more seizures followed the administration of the drug and then they ceased, and he fell soundly asleep. April 11, 1875—He had twelve seizures between nine last night and eight this morning. The intervals, long at first, are diminishing steadily. Seizures as follows: a faint cry continued in a shriller tone through the tonic spasm; the eyeballs rolled upwards and to the right, the mouth opening widely; then the muscles of neck, finally muscles of trunk and limbs are convulsed; arms in the tonic spasm extended fully and rigidly; the tonic spasm lasts fifteen seconds, the clonic spasms forty-five seconds. He had eight seizures between 8 A.M. and 2 P.M., ʒss of chloral having been injected at the former hour, with the effect of lessening the number of seizures and lengthening the intervals; at 2 P.M. a pint of beef-tea, 2 ozs. brandy, and ʒss of chloral hydrate were injected; four seizures up to 6 P.M., when they increased perceptibly in severity and number, five seizures occurring between 6.0 and 8.30, when ʒss of chloral was administered with a pint of milk, after which the seizures ceased, patient falling asleep. June 17, 1874, he had about this time several batches of fits, in some the attacks numbering as many as seventeen and nineteen; nitrite of amyl was exhibited without much effect. December 24—The fits recurred last night, and patient had seven in rapid succession, ʒss of chloral administered—fits ceased.

CASE 6.—T. K——, æt. 6, was admitted on June 8, 1874. Has been epileptic from birth. At two years old he had a kick from a horse. Fits occur in batches, with intervals of a fortnight and upwards; from time of admission until August 10 he had one or two slight seizures; afternoon of August 10 had a series of very rapid and severe seizures lasting about an hour and a half, the convulsions being most marked on left side; nitrite of amyl and chloroform inhalations were tried, the former without effect, the latter succeeding as long as the inhalation was continued; finally convulsions ceased. October 28—This evening a severe fit occurred at seven, passing into clonic spasms, which continued for fifteen minutes, then intermitted for five minutes, when a second fit occurred, the clonic spasms which continued till 8.20. At 7.30 nitrite of amyl was inhaled with the effect of producing at first a slight decrease in the severity of the symptoms, but the attack soon resumed its former intensity, and nitrite of amyl was administered repeatedly in five minim inhalations with absolutely no effect. At 8.10 an injection of fifteen grains of chloral was administered by the rectum,

convulsions continuing for five minutes as before, when they began to decrease perceptibly in intensity. At 8.20 all spasms had ceased save in the right arm, where slight twitchings of the flexors were visible for five minutes or so longer. A violent hiccough now commenced, which lasted for about two minutes, then gradually subsided, and twenty-five minutes after the exhibition of the chloral the patient was asleep, and continued to sleep the whole night through without any return of fits. January 17, 1875, became convulsed at 11.0 to-day ; seen by reporter at 11.30—clonic spasms of great severity affected nearly the whole body ; muscles of face very generally affected, distorting the child's face to an unusual extent ; respiration consisted in a series of short jerks and interrupted by hiccough. The convulsions seemed to observe a certain rhythmic increase and decrease in severity ; the body was bathed in perspiration ; pulse uncountable ; hiccough ended in the ejection of a quantity of glairy mucus from the stomach. At 11.35 an injection of fifteen grains of chloral in 2 ozs. of water was passed up the rectum ; at 11.50 a return of the vomiting took place and nearly asphyxiated the boy, as some of the glairy mucus was ingurgitated into the larynx. He was rolled on to his side, and his mouth emptied ; respiration had now ceased ; his face was livid ; no sign of life save a faint thready pulse and a very fine tremor of the muscles of the trunk ; artificial respiration was commenced and carried on for about five minutes, when a faint sighing inspiration took place, and natural respiration was re-established with continued sighing, and irregular as to rhythm for some minutes. 12.15 P.M., the tremor of the muscles and rolling of eyeballs passing off. 12.45 P.M., patient was left sleeping soundly, all convulsions having ceased, and the pulse and respiration both slower. March 17, 1875— A seizure occurred at 9.30 A.M. ; it was unilateral and lasted two or three minutes. After an interval of ten minutes a second fit occurred, the tonic spasms of which gave place to clonic convulsions of great severity, which lasted for an hour and eight minutes, during which time patient was kept without chloral, designedly to see whether the attack would terminate spontaneously. As he was becoming livid, and this respiration seriously embarrassed fifteen grains of chloral were injected into the rectum, and in five minutes he was asleep, all convulsions having ceased.

CASE 7.—J. L.——, æt. 24, was admitted on October 24, 1868. Is a confirmed epileptic, in rather delicate health. February 7, 1875—Patient had nineteen fits yesterday up to 10.0, and was passing into the status epilepticus when he had ʒss of chloral, and slept without recurrence of convulsions until 2.15, when a seizure occurred, followed by others at 2.50, 3.10, 3.25, 3.30. ʒss chloral repeated ; a sixth fit at 3.35, a seventh at 4.0, when patient went to sleep, and did not awake until 7.30, when a slight fit occurred, followed by a very slight one at 8.15 ; no others afterwards.

CASE 8.—M. C——, admitted on September 13, 1869. Epileptic from infancy. July 23, 1874, he was seized with a fit, which was rapidly followed by others, and he eventually died on July 25, 1875, having pneumonia of the right side, and having suffered 530 distinct seizures since the commencement of the attack in a period of forty-eight hours. Chloral hydrate was exhibited over and over again, but without effect, further than lessening the number of seizures, or rather prolonging the intervals for a

short time.   Inhalations of chloroform and nitrite of amyl were also exhibited, but without effect; succus conii was also tried in ℥ss doses, the first of which was followed by a lengthening of the intervals between seizures from five to twelve minutes.   Patient was supported by beef-tea, milk, brandy, &c. The post-mortem revealed the firm, plump brain of chronic epilepsy, which seemed to bulge out from beneath the dura mater when the latter was opened. The sinuses and vessels were gorged with blood and coagula; the right vertebral artery was very much smaller than the left one, being about one-third of its calibre. No other marked peculiarities were found; the carotids were equal in size.

CASE 9.—J. H. T——, æt. 22, was admitted on February 6, 1873. He is a confirmed epileptic, in moderate bodily health. On September 10, 1874, he had eighteen fits up to 10 P.M.; bowels were freely opened by an injection; at 7 P.M. he had ℨss of chloral; five seizures occurred between 7 and 10; a second dose of forty-five grains was given him, when he fell soundly asleep, and had not a single convulsion afterwards.

CASE 10.—J. G——, æt. 40, was admitted on September 25, 1872. A confirmed epileptic in good bodily condition. On April 12, 1875, had fifteen seizures from 6.0 to 10 P.M.; chloral hydrate to the extent of forty grains was then given him, seemingly without effect, the convulsions recurring at short intervals all night. April 13, 10 A.M., forty grains of chloral were administered. Loud râles audible all over the chest; patient remained profoundly comatose between the fits. 2.45 P.M., chloral has been without effect; pulse 136, weak; a pint of beef-tea, 2 ozs. whisky, and ℨj of chloral were given by the rectum. 5 P.M., no improvement followed last administration, and patient died in the twenty-seventh seizure since 6 A.M.

CASE 11.—G. F. T——, a congenital imbecile and epileptic from infancy, in fair bodily condition, æt. 15, was admitted on July 15, 1867. He had a fit in the afternoon, and had four after going to bed; at 12.30 had a fifth seizure very severe in character, and followed by the most violent movements of his limbs; twenty grains of chloral sent him to sleep, and caused the jerking movements to cease.

CASE 12.—R. C——, æt. 53, was admitted May 2, 1862. Is a confirmed epileptic. Had eleven seizures on the morning of April 20, 1875, up to 1.0, when he was found lying comatose, not having recovered his consciousness after the last four or five attacks; body bathed in perspiration; respiration free; forty grains of chloral given him by enema; two more seizures subsequent to exhibition of chloral, followed by sound sleep and cessation of convulsions.

CASE 13.—F. S——, æt. 49, readmitted November 1, 1866. Was a resident three years previously, and has been epileptic from his childhood. April 22, 1871, had a series of convulsions, and forty grains of chloral were given him; convulsions ceased.

CASE 14.—J. S——, æt. 69, readmitted on November 9, 1866. A senile dement with occasional attacks of excitement. Has suffered previous repeated attacks of mania; age on first admission 40.   January 4, 1873, he had an attack of hemiplegia, from which he never recovered much. October 25, 1874, had an epileptic fit in the garden this morning; arms and legs affected on both sides, but only left side of face; he remained semi-conscious until next morning. March 14, 1875, was found by night attendant

in a severe fit at 10 P.M.; this attack was followed by three others not quite so severe; bowels having been cleared out by enema, forty grains of chloral were given; the seizures recurred of a slighter degree of severity, but as rapidly as before until 12.0 midnight, when a second dose of thirty grains was administered, after which two slight fits occurred at longer intervals, and then the convulsions ceased altogether.

CASE 15.—E. G. S——, admitted July 25, 1874. Has been epileptic for last ten years. On November 11, 1874, retired to bed in usual condition, and was found by the nurses, who were awakened by her loud snoring at 3.5 A.M. of November 12. Was seen by Dr. Major, who found her in the status epilepticus, perfectly comatose, cyanosed ; pulse 130; skin perspiring; breathing embarrassed, loud mucous râles being audible all over the room. Fit succeeded fit without more than a few seconds interval; forty grains of chloral were given by the rectum; seizures continued as before for four or five minutes, then there was a perceptibly longer interval; another attack, a longer interval; ten minutes after the administration of the drug the fits ceased altogether.

CASE 16.—L. G——, admitted September 23, 1873, suffering from mania. October 27, 1874, she had the first epileptic attack since admission, and first she was ever known to have; no paralysis before or after the fit. November 6, was noticed to drag the left foot in walking and to have loss of power in left hand. November 7, stumbled and fell, and had two distinct epileptic fits. February 16, 1875, she was found in a fit at 7 A.M.; fit followed fit in rapid succession, and at 8.45 she was in the status epilepticus; pulse 120; face deeply cyanosed; profoundly comatose, and utterly unable to swallow. An enema of thirty grains of chloral was given, after which the fits occurred as follows : first at 9 A.M., second at 9.5 A.M., third at 9.30 A.M., fourth at 2.35 P.M., fifth at 6.35 ; after which she remained free from convulsions. May 20, to-day she had a return of epileptic seizures, and after several had occurred in rapid succession, thirty grains of chloral were given, and no further fit took place, patient falling into a sound sleep in a few minutes.

CASE 17.—H. B——, admitted November 2, 1872 ; has been epileptic for three and a half years. June 22, 1875—convulsed this afternoon, and had eighteen seizures between dinner time and 8 P.M. He is partly conscious between the attacks and able to swallow; pulse 128 ; breathing tolerably free in the intervals. At 8 P.M. had forty grains of chloral, and fell asleep in a few minutes, and remained without fits until 1 A.M., June 23, when the seizures recurred. Had forty grains of chloral at 2.30 A.M., but the seizures continued at intervals until 8.50, when a third dose of forty-five grains was administered ; he fell asleep in five minutes, and slept until 1.30, when he awakened. He began to have seizures again at 4.15 P.M., and had twenty in all up to 6 A.M., June 24, no chloral having been given. At 6.10 A.M. forty-five grains of chloral were injected and patient slept soon after, and had no further fits, awakening occasionally to take food. This patient has had several recurrences of frequent seizures, which have been in every case arrested speedily by a single dose of forty-five grains.

The power possessed by chloral to control the number and severity of epileptic convulsions will surely be admitted after

the perusal of the foregoing cases, which, did space permit, might be multiplied greatly from the Asylum Case-Books. There is no necessity for a lengthened analysis of the examples quoted, but it may be observed that in the great majority of instances a marked and almost immediate effect was produced by the administration of a full dose of the drug. The interval between the seizures was lengthened, and the convulsions were reduced in intensity, and finally abolished. The pulse, at first increased in tension, became slower and softer to the touch; respiration became slower and unembarrassed. Occasionally a case was met with in which chloral seemed almost powerless, although pushed as far as three doses of $\mathfrak{z}$j at half-hour or three-quarter-hour intervals. Two of these cases have been given—namely, Cases 7 and 10—in which the exhibition of chloral was of no effect, save in producing a slight temporary lessening of the number of attacks, but in these cases other modes of treatment were equally ineffectual. No explanation can yet be offered as to the cause of failure in these cases, unless it be that there existed in them a specific antagonism to the drug, which certainly did not produce in them any of its usual physiological effects, such as slowing and lowering the tension of the pulse, reducing the temperature and frequency of the respirations, and producing sleep. Again, it may perhaps depend upon the eccentric origin of the stimulus giving rise to the seizures.

We have now to consider the physiological action of the drug, and how this action is applied to remedy the convulsive state. The earliest writers on chloral, notably Drs. B. W. Bennett and Personne, entertained the idea that chloral acted by being split up into chloroform and formiate of soda or potass. Personne obtained chloroform from the blood of animals poisoned by chloral, but his distillation was carried on at a much higher temperature than that of the human body, and it is urged that the formation of chloroform may have been influenced by the great heat. Then Hammarsten Rajewsky and Amory found that in dogs, deeply poisoned by chloral, chloroform could not be detected in either the expired air or the blood, whereas when chloroform was used it could be detected in the breath. These and other experiments proved

conclusively that chloral mixed with blood at ordinary tem-
peratures does not suffer change. The effects of chloral are,
though generally resembling those induced by chloroform,
not at all identical; equal doses of the drug produce dif-
ferent symptoms, the sleep induced by chloral being more
prolonged, and the anæsthesia induced by chloroform much
more intense. The physiological action of chloral in mode-
rate doses may be summed up as follows:—It produces
sleep, muscular relaxation, lessening of the tension of the
pulse (after a primary increase), lessening of the number of
respirations, and lowering of the temperature.

The next point is, how is this action applied to epilepsy,
and first, what is epilepsy? The sum of our exact knowledge
on the subject of the group of phenomena called epilepsy is
but small. Dr. Hughlings Jackson, whose able and patient
investigations in this direction promise a harvest of rich
results, defines epilepsy as being a sudden, occasional, exces-
sive, and rapid discharge of grey matter of some part of the
brain.

The epilepsies we have been considering in this paper may
be divided pathologically into three classes: first, the
ordinary simple epilepsy; second, the epilepsy accom-
panying a hæmorrhage; third, the epilepsy occurring in
the course of general paralysis. In the first class we must
suppose a permanent condition of mal-nutrition, resulting in
the excessive development of nerve force which periodically
reaches a state of tension when it discharges itself.

In the second class, a more or less normal condition of
nutrition exists, but a sudden and unusual stimulus is applied
in the shape of effusion of blood and laceration of nerve
tissue, and the irregular discharge ensues.

In the third class, where sudden epilepsies occur, in ad-
vanced general paralysis, we certainly have perverted nu-
trition as well as degeneration, but the difficulty is to know
why this should produce explosions in one case and not in
another.

Whatever may be the remote cause acting in these dif-
ferent classes of convulsions, it may be seen that chloral has
a very marked influence over them all. We have seen that

the action of chloral is primarily exerted on the grey matter of the hemispheres, producing sleep, hence we may infer that in all these cases there exists a common abnormal or perverted condition of the grey matter, to which the convulsions are immediately due, and over which chloral exercises a specific influence. How this influence is exerted we know not; it may be directly by chemical inter-action between the elements of the chloral on the one hand, and those of the grey matter on the other, resulting in the elaboration of a more stable tissue than that previously existing, or indirectly through the action of chloral on the arterial system, by paralysing the vaso-motor nerves; or finally, by its action on the heart itself. Whatever be the manner of its action, of its result there can be but little doubt. Since it began to be systematically used by the writer in all cases of repeated epileptic seizures, the deaths from this *cause* have diminished in a most notable degree, both in his experience, and that of his colleagues who have exhibited it to their patients. As to the mode of its administration, it may be either given by the mouth, or, if the power of swallowing be lost, by the rectum, dissolved in water, and with or without beef-tea, brandy, &c. It acts quite as satisfactorily by the rectum, and, if anything, rather more rapidly than when given by the mouth. Whenever it has been injected subcutaneously, it has, in reporter's experience, caused considerable sloughing in the neighbourhood of the injection. Finally, as to the dose. For an adult a single dose of ʒj is quite safe, and much more effectual than ʒss doses divided by an interval. It may be repeated in an hour if necessary, but the first dose usually causes the convulsions to cease, and plunges the patient into a profound sleep.

LARYNGOSCOPIC OBSERVATIONS IN GENERAL PARALYSIS.

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HESITANCY and imperfection in the enunciation of articulate sounds—in other words, difficulties of speech expression—are of such common occurrence in patients suffering from general paralysis, that it is somewhat strange that they have not received more attention. Beyond a short and most interesting article in 'The British Medical Journal' (July 19, 1875), by Dr. Voisin, Physician of La Salpêtrière, I am unacquainted with any author who has attempted to differentiate the troubles of speech of general paralytics, and to account for their causation. It was with the greatest pleasure, therefore, that early in the spring of this year I accepted the invitation of Dr. Crichton Browne to make some investigations under his guidance of this interesting subject, and the abundant wealth of material at his command offered a rich field for observation. In Dr. Voisin's article an endeavour is made to find causes for the defects in enunciation—or, as is erroneously said, of language—in the various pathological alterations in the brain or medulla of general paralytics, and this is done with very considerable success, by bringing to bear a large experience in the post-mortem room on generally accepted physiological facts.

The present series of observations, on the other hand, is confined wholly to the living, and comprises the results

obtained from an ordinary laryngoscopic observation of fifty well-marked cases of general paralysis at one and the same time under treatment in the West Riding Asylum. The observations were made at a time when there were 1,424 patients in the Asylum, of whom 707 were males and 717 females. There were about 25 other patients labouring under the disease at the time in the Asylum, all of whom I examined. These have been omitted because the malady was complicated with other diseases, or because the examination was for various reasons not perfectly satisfactory. The number has thus been reduced to 50, of whom 37 are males and 13 females. The appended table gives all the information which could be derived from an ordinary examination of any patient with the laryngoscope, and the various facts are arranged in the order of sequence in which each would present itself to the observer. The points considered are as follows :—

1. Utterance—This includes, it is true, some points which do not depend on local neuro-muscular but on cerebral changes. It has been thought better, however, to give the result of actual observation of this condition irrespective of cause.

2. Quality of voice.

3. Oral power—comprising control over lips and muscles of the jaw.

4. Lingual power—that is, power of protrusion of tongue and condition of that organ.

5. Condition of pharynx, including velum, uvula, &c.—
  a. Reflex sensibility.
  b. Muscular condition.—This refers especially to whether there was relaxation of these parts.
  c. Vascular condition of pharynx.

6. Condition of larynx—
  a. Position of epiglottis.
  b. Vascular condition
    1. Of mucous membrane generally.
    2. Of vocal cords.
  c. Nervo-muscular condition of vocal cords.

In addition, as a matter of interest, the presence of a

hæmatoma, or any other fact appearing to be worthy of note, is added.

The cases were examined indiscriminately as to length of residence in the Asylum or of stage of disease, though, for the sake of convenience, they will be arranged according to the progress of the malady as afterwards given to me by Dr. Crichton Browne. The initiative stages of a laryngoscopic examination, the opening of the mouth, and the taking hold of the tongue,[1] were, from difficulties caused by the disease, by no means easy; but these accomplished, the mirror was in all cases well tolerated, and not one case of retching due to reflex irritation occurred. Indeed, to those unaccustomed to laryngoscopic work, the process seemed most easy, and it was only when non-paralytic patients suffering from other laryngeal disease were examined, that medical friends (unpractised in the use of the instrument), who were present at these observations, discovered that the tolerance with which the mirror is borne by general paralytics is due to diminution of sensibility of the pharynx. Almost every case would bear not only the mirror, but direct irritation without reflex movement. In all there was great relaxation and flabbiness of the velum and frequently hyperæmia. The number of cases in which the epiglottis was unduly pendant was far in excess of the average of a corresponding number of normal cases, this condition being due to over-relaxation of the glosso-epiglottic folds. There was less inequality in the co-ordination of the laryngeal muscles than would perhaps at first be supposed. In the majority of cases where there was impediment, it was of the nature of unilateral impairment of approximative power. The vocal cords were also frequently seen to be distinctly wanting in tensor power. In no case was there any symptom of dyspnœa.

[1] It has been noted in the Tables that many patients raised their hands to assist in protrusion of the tongue. This they did on being told to put it out, and expresses their own sense of inability to perform this simple act.

No. of Case	Initials and sex	Age on admission	Date of admission	Stage of disease	Utterance	Voice	Oral power	Lingual power	Pharynx Reflex sensibility	Pharynx Muscular condition	Pharynx Vascular condition	Larynx Position of epiglottis	Larynx Vascular condition of mucous membrane generally	Larynx Vascular condition of vocal cords	Nervo-muscular condition of vocal cords	Remarks
1	F. P. Male	29	March 25, 1875	First	Good	Slightly coarse and thick	Good	Well protruded	Diminished	Relaxed	Congested	Low	Congested	Normal	Good	
2	I. M. Male	43	July 28, 1875	Early part of second	Stammering	Hoarse and thick	Good, lips tremulous	Well protruded, slightly tremulous	Normal	Relaxed	Congested	Upright	Congested, much frothy mucus	Normal	Good	
3	J. H. F. Male	41	March 20, 1875	Second	Good	Loud and coarse	Good	Good	Diminished	Relaxed	Slightly congested	Low	Congested	Normal	Normal	
4	C. L. Male	37	July 17, 1875	Second	Hesitating	Hoarse and thick	Good	Tremulous	Diminished	Relaxed	Congested	Upright	Normal	Normal	Diminished power in approximation, especially of left	
5	J. F. Male	30	Oct. 18, 1873	Second	Hesitating and with long interruptions	Thick and somewhat indistinct	Fair	Fair, protrusion, but very tremulous	Diminished	Relaxed	Normal	Upright	Normal	Normal	Normal	Congenital over-riding of arytenoids in phonation

No.	Name and Sex	Age	Date	Stage												
6	D. M. Male	39	Sept. 3, 1874	Second	Hesitating	Coarse	Diminished	Diminished	Greatly diminished	Normal	Much congested	Low	Congested	Normal	Normal	
7	C. W. Male	41	Nov. 4, 1872	Second	Great hesitation	Hoarse	Tremulous	Protrusion, jerky and uncertain		Relaxed	Congested	Upright	Congested	Normal	Quivering, and hesitating	
8	W. H. Male	39	April 30, 1875	Second	Good, but hesitation of thought and forgetfulness during speech	Good	Good	Good and steady	Fair	Relaxed	Congested	Upright	Congested	Normal	Normal	
9	M. A. W. Female	33	Dec. 20, 1872	Second	Hesitating and frequent repetition of words and syllables	Good	Fair	Fair; hand raised to assist	Greatly diminished	Relaxed	Congested	Upright	Normal	Normal	Normal	
10	G. P. Male	49	Jan. 18, 1875	End of second	Thick; words slurred over in last syllables	Hoarse	Fair	Diminished; tongue tremulous and lolling	Greatly diminished	Relaxed and flabby	Normal	Low	Normal	Normal	Normal	
11	S. H. Male	39	Jan. 4, 1875	End of second	Stammering	Hoarse and thick	Good	Good	Normal	Slightly relaxed	Congested	Upright	Congested	Normal	Normal	Hæmatoma of left ear
12	H. E. Male	45	June 13, 1873	End of second	Mumbling and slurring	Hoarse	Fair	Fair, slightly tremulous	Fair	Very flabby	Congested	Low	Congested	Normal	Normal	Hæmatoma of left ear
13	J. W. Male	40	Aug. 29, 1874	End of second	Commencing well; hesitates frequently during sentences	Normal	Good	Good and steady	Much diminished	Slightly relaxed	Normal	Low	Normal	Normal	Normal	

No. of Case	Initials and sex	Age on admission	Date of admission	Stage of disease	Utterance	Voice	Oral power	Lingual power	Pharynx			Larynx				Remarks
									Reflex sensibility	Muscular condition	Vascular condition	Position of epiglottis	Vascular condition of mucous membrane generally	of vocal cords	Nervo-muscular condition of vocal cords	
14	J. J. S. Male	63	Nov. 10, 1874	End of second	Good	Coarse	Good	Fair, but tremulous	Diminished	Relaxed	Congested	Upright	Congested	Normal	Want of power in left vocal cord	
15	R. F. Male	38	Jan. 9, 1875	End of second	Hesitating and stammering	Thick	Great tremor	Diminished with tremor	Fair	Relaxed	Congested	Low	Congested	Normal	Normal	
16	T. G. Male	44	April 24, 1874	End of second	Hesitating	Hoarse	Fair	Fair, hut protrusion slightly to left	Much diminished	Relaxed	Considerably congested	Normal	Much congested	Congested	Unequal lateral action, decided want of power in left adductor	
17	E. G. Male	42	Oct. 30, 1873	End of second	Hesitating	Hoarse	Slight labial tremor	Diminished tremulous	Much diminished	Relaxed	Much congested	Low	Much congested	Slightly congested	Normal	
18	H. W. Female	44	July 28, 1875	End of second	Indistinct, without hesitancy	Thick and hoarse	Tremulous	Greatly diminished; hand raised to assist protrusion	Much diminished	Relaxed	Congested	Normal	Congested	Normal	Great want of adductive power of left vocal cord	Irregularity of pupils

19	J. H. Male	41	May 6, 1875	Begin-ning of third	Hesitating; in counting skips figures	Coarse	Tremulous		Much dimin-ished	Relaxed	Con-gested	Low	Con-gested	Slightly con-gested	Want of prompt-ness approxi-mation	Hæmatoma of left ear
20	J. L. Male	47	March 15, 1875	Begin-ning of third	Hesitating	Coarse but loud	Slight labial tremor	Fair and prompt	Dimin-ished	Relaxed	Con-gested	Slightly pendu-lous	Con-gested	Slightly con-gested	Want of prompt-ness in action	
21	W. H. Male	42	July 18, 1874	Begin-ning of third	Stammer-ing	Harsh and coarse	Fair	Fair, with slight tremor	Greatly dimin-ished	Relaxed	Con-gested	Low	Con-gested	Slightly con-gested		
22	W. W. Male	46	May 4, 1874	Begin-ning of third	Hesitancy as of loss of memory in middle of sentences	Thick	Mode-rate	Moderate	Dimin-ished	Relaxed	Con-gested	Quite ob-structing view of vocal cords	Con-gested	Normal	—	Signs of syphilitic ulceration of tongue
23	G. S. Male	48	April 11, 1874	Begin-ning of third	Great hesi-tation and slurring of articu-lation	Thick	Dimin-ished	Enfeebled	Much dimin-ished	Much relaxed	Much con-gested	Low	Con-gested	Normal	Adduc-tive power dimin-ished	
24	E. I. Female	37	Dec. 28, 1874	Begin-ning of third	Hesitating; drawling with much pro-longation of last syllables	Rather but nearly normal	Fair	Fair	Much dimin-ished	Relaxed	Con-gested	Low	Con-gested	Normal	Normal	
25	C. T. Female	23	Oct. 5, 1874	Begin-ning of third	Mumbling	Slightly hoarse	Dimin-ished	Only partial protrusion possible, tremulous and slightly to left side	Much dimin-ished	Relaxed	Con-gested	Low	Slightly con-gested	Normal	Normal	Hæmatoma of left ear

No. of Case	Initials and sex	Age on admission	Date of admission	Stage of disease	Utterance	Voice	Oral power	Lingual power	Pharynx			Larynx				Remarks
									Reflex sensibility	Muscular condition	Vascular condition	Position of epiglottis	Vascular condition of mucous membrane generally	of vocal cords	Nervo-muscular condition of vocal cords	
26	M. L. Female	36	June 26, 1873	Beginning of third	Mumbling; imperfect articulation	Thick	Much diminished	Much diminished; hand raised to assist in protrusion	Fair	Normal	Very slightly congested	Low	Normal	Normal	Normal	
27	J. D. Male	33	Oct. 17, 1874	Beginning of third	Uncertain, sometimes clear, at others inarticulate; slight hesitancy	Thick	Fair	Enfeebled	Diminished	Relaxed	Congested	Low	Congested	Normal	Normal	
28	G. S. Male	43	Dec. 19, 1873	Third	Good, but tremulous	Normal	Fair, with labial tremor	Fair with tremor	Diminished	Relaxed	Congested	Low	Congested	Normal	Normal	
29	J. T. Male	36	Oct. 3, 1873	Third	Very hesitating and drawling of last syllables	Normal	Fair	Diminished with tremor	Diminished	Relaxed	Congested; much frothy mucus	Low	Congested	Normal	Normal	

30	B. F. Male	38	March 31, 1874	Third	Great want of sharpness; prolongation of last syllables	Coarse	Dimin-ished	Much dimin-ished with tremor	Dimin-ished	Relaxed	Con-gested	Upright	Con-gested at posterior part	Slightly con-gested	Left vocal cord immobile midway between ad- and abduction	Hæmatoma of left ear
31	F. A. Male	40	Dec. 8, 1873	Third	Constant inarticulate mumbling	Loud and coarse	Dimin-ished	Dimin-ished	Much dimin-ished	Relaxed	Much con-gested	Low	Con-gested	Con-gested	Normal	
32	O. O. Male	33	July 20, 1874	Third	Good	Hoarse	Dimin-ished, great labial tremor	Dimin-ished	Much dimin-ished	Relaxed	Much con-gested	Normal	Con-gested	Slightly con-gested	Normal	Hæmatoma of both ears. Patient excitable and refractory
33	M. H. Male	39	April 13, 1873	Third	Fair	Coarse	Fair, slight tremor	Fair, slight tremor	Fair	Normal	Slightly con-gested	Low	Slight conges-tion	Normal	Normal	Hæmatoma of both ears
34	T. H. Male	42	June 19, 1875	Third	Want of clearness; words run into each other	Coarse droning	Fair, slight tremor	Fair, slight tremor	Dimin-ished	Relaxed	Con-gested	Low	Con-gested	Normal	Normal	
35	W. C. Male	60	April 22, 1875	Third	Great hesi-tation	Thick	Much dimin-ished, labial tremor	Much dimin-ished with tremor	Absence of reflex sensi-bility on irri-tation	Much relaxed	Con-gested	Normal	Con-gested	Normal	Great want of pre-cision and prompt-ness in approxi-mation	

No. of case	Initials and sex	Age on admission	Date of admission	Stage of disease	Utterance	Voice	Oral power	Lingual power	Pharynx			Larynx				Remarks
									Reflex sensibility	Muscular condition	Vascular condition	Position of epiglottis	Vascular condition: of mucous membrane generally	of vocal cords	Nervo-muscular condition of vocal cords	
36	J. B. Male	36	Jan. 26, 1875	Third	Good	Rather hoarse	Good	Fair	Diminished	Relaxed	Congested	Upright	Congested	Normal	Normal	General relaxation of muscles of larynx, the ventricular bands and vocal cords being very lax
37	L. I. Male	32	June 15, 1875	Third	Hesitancy and drawling	Hoarse	Fair	Fair; protrusion to right side	Diminished	Relaxed	Congested	Upright	Slightly congested	Normal	Want of adductive power of left vocal cord	
38	F. S. Male	40	April 16, 1875	Third	Stammering and stuttering; hesitation during sentences	Thick	Fair	Fair with tremor	Diminished	Relaxed	Congested	Upright	Slightly congested	Normal	Want of adductive power of right vocal cord	

39	A. S. Male	29	Dec. 19, 1874	Third	Slow, hesitating; drawls and slurs over last syllables; words omitted in some sentences	Hoarse	Fair	Protrusion jerky and tremulous	Much diminished	Relaxed	Con-gested	Low	Con-gested	Normal	Normal	
40	T. G. Male	53	Aug. 20, 1874	Third	Hesitating	Hoarse	Dimin-ished	Most feeble hand raised to assist pro-trusion	Much dimin-ished	Very re-laxed	Con-gested	Low	Con-gested	Normal	Normal	
41	B. A. Male	47	Oct. 18, 1873	Third	Hesitating, mumbling and almost in-articulate	Thick and hoarse	Dimin-ished	Very feeble	Much dimin-ished	Relaxed	Con-gested	Low	Con-gested	Normal	Normal	
42	F. H. Female	26	May 5, 1874	Third	Great hesi-tation and nervous-ness in articula-tion	Hoarse	Fair	Good, but very tre-mulous	Dimin-ished	Very re-laxed	Much con-gested	Rather pendu-lous	Con-gested	Normal	Slight want of approx-imative power of left vocal cord	Very emo-tional
43	M. M. Female	40	Aug. 11, 1873	Third	Fairly good	Normal	Good	Good, but tremulous	Dimin-ished	Relaxed	Slightly con-gested	Low	Normal	Normal	Normal	
44	M. B. Female	45	May 5, 1873	Third	Indistinct and drawling	Harsh, shrill, metallic	Great tremor	Feeble, with great tre-mor	Dimin-ished	Relaxed	Con-gested	Normal	Con-gested	Normal	Very ir-regular and jerky	
45	J. J. Male	25	Dec. 8, 1874	Third	Hesitating	Thick	Fair	Fair; tongue large and indented with teeth marks	Dimin-ished	Relaxed	Con-gested	Normal	Con-gested	Con-gested; right vocal cord thick-ened	Great want of power of right vocal cord	

No. of case	Initials and sex	Age on admission	Date of admission	Stage of disease	Utterance	Voice	Oral power	Lingual power	Pharynx			Larynx				Remarks
									Reflex sensibility	Muscular condition	Vascular condition	Position of epiglottis	Vascular condition of mucous membrane generally	of vocal cords	Nervo-muscular condition of vocal cords	
46	E. S. Female	39	Dec. 21, 1872	Third	Hesitating, drawling and repetition of words	Hoarse	Feeble	Very feeble and tremulous, hand raised to assist protrusion	Diminished	Relaxed	Rather congested	Normal	Normal	Normal	Normal	Very emotional
47	E. S. Female	43	July 20, 1875	Third	Rather mumbling	Clear	Fair	Fair; slight tremor	Fair	Relaxed	Slightly congested	Low	Slightly congested	Normal	Deficient adductive power Feeble	
48	E. E. Female	34	Nov. 17, 1871	Third	Stammering and stuttering with dribbling of saliva	Thick and hoarse	Feeble	Feeble and tremulous; hand raised to assist protrusion	Greatly diminished	Relaxed	Congested; much frothy mucus	Low	Congested	Normal	Feeble	Very emotional
49	C. B. Female	43	May 27, 1873	Advanced in third	Drawling; imperfect articulation with salivary dribbling	Thick	Feeble, great labial tremor open mouth rather to left side	Feeble, tongue large, lolling and quivering	Greatly diminished	Relaxed	Congested	Low; very congested	Congested	Slightly congested	Feeble	Very emotional
50	E. R. Female	30	Oct. 11, 1869	Advanced in third	Inarticulate	Hoarse and quavering	Fair	Fairly protruded; but large and tremulous	Greatly diminished	Relaxed	Congested; much frothy mucus	Low; very congested	Congested	Normal	Feeble	Very emotional

The following facts may be considered established as the result of these observations :—

First. That in general paralysis of the insane the power of utterance is impaired proportionately to the advance of the disease, being good in the first and early part of the second stage, bad in the latter part of the second stage, and very bad in the third stage.

Secondly. That the voice becomes thick and coarse at a very early period, and that it is not infrequently hoarse.

Thirdly. That the power of protrusion of the tongue is diminished from the commencement of the disease, and becomes gradually more and more impaired as it progresses.

Fourthly. That the reflex excitability of the pharynx is markedly diminished from the beginning of the disease.[1]

Fifthly. That there is generally relaxation of the velum and of the mucous folds and muscles of the larynx, with hyperæmia of the pharynx throughout the whole course of the disease.

Sixthly. That there is impairment of tension and of co-ordinate action in the vocal cords, unaccompanied by any distress of respiration. This last fact, coupled with the absence of reflex activity, suggests that it is the superior laryngeal nerve rather than the inferior which is affected.

The observations recorded in this paper point unmistakably to the involvement of motor and sensory nerves and their centres, as well as of the sympathetic system in the pathological process in which this still obscure and fatal malady essentially consists ; and are therefore, I believe, in perfect harmony with the most recent results of microscopic research, and of clinical investigation. I trust that they may serve to throw some light upon the nature of general paralysis, and upon the causation of some of its most common and pronounced symptoms.

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[1] The reflex sensibility of the larynx was not uniformly tested, but judging from a few cases in which the epiglottis was touched, there is probably diminished sensation of the mucous membrane of the larynx as well as of the pharynx.

NOTE ON CHRONIC MANIA.

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'CHRONIC mania,' says Bucknill, in one of those vivid descriptions that give a peculiar charm and value to his writings, 'represents the rudderless and shattered state of the vessel after the tornado of raving madness has swept by. The wreck is left in every variety of condition; sometimes with sail enough to keep her head to wind, sometimes she lies upon the waters a log in the helpless state of consecutive dementia.' Those wrecks of humanity depicted in the accompanying illustration have passed through the tornado, and have come out of it hopelessly damaged, and yet not so utterly scathed and broken as are many other goodly vessels that have encountered the same tropical hurricane of passion; they have lost helm and compass, and must drift henceforth in circular voyages without port or purpose, but they are not altogether derelict of reason, nor are they threatened with speedy engulfment. They belong, in fact, to the mildest variety of chronic mania. In them the acute attack of insanity with which their mental troubles commenced, has ended in disorder rather than enfeeblement of intellect. Their perceptive faculties are as lively as ever. They notice all that takes place around them. They apprehend whatever is said to them, and reply with more than ordinary celerity. Their memories have a strong grasp on the past, except that portion of it which was covered by the primary stage of their mental malady, and they are quick to recognise, classify, and name the persons and objects that are brought before them. Their acquirements have not gone from them. They

can read, write, or cipher nearly as well as they ever did, but they are incapable of continuing any exercise of the kind for more than a few minutes consecutively.

No superficial nor distinct signs of mental decay are to be detected in them, and yet they are as much incapacitated as if they were demented. They have lost control over their mental processes. They cannot pursue any train of thought. Like the carnal sinners of Dante's second circle, they are ' whirled round and dashed amain with sore annoy ' by stormy blasts of emotion and tyrannous gusts of ideas. They can no longer regulate the inner life, and that is no longer in harmony with the outer environment. They are not so much weak as wild minded. They are not so much fatuous as fantastic. The proud and compact phalanx of intellect is almost unthinned, but it has been converted into an unruly mob. The steady tread of rational progression is possible to them only for a moment, and has given place for the most part to the hurried shuffling and confused scrambling of mental tumult. Their minds are in some respects more active than they were wont to be in health, and a copious flow of ideas passes through them; but the activity is morbid, and the ideas are incongruous and apparently disconnected. Then the gift of reticence is lost to them. They cannot think silently, or with only faint external indications of the current of thought within; but they must babble out all their delirious conceptions, or at least as much of them as articulation at its quickest is capable of keeping pace with. Their conversation is more incoherent than their ideas, probably because thought distances expression, and because utterance can only be given to snatches here and there of the fast-coming fancies that course through the brain. In listening to the rambling, inconsequential diatribes of chronic maniacs, one is often able to trace out some hidden or submerged connection between portions of them that might at first seem to have no relationship ; one is often able to perceive that the discourse is not a full reflex of thought, but that many ideas are elided in expression, while others, comparatively remote from each other, are made confluent. Hence in some measure are to be explained the strange combinations,

the abrupt and unexpected transitions that occur in their endless monologues. Beyond this, however, there is of course great irregularity in the succession of the ideas themselves, which arise not in accordance with ordinary laws of association, but in new and grotesque groups, which are the direct offspring of cerebral excitement. All the psychical processes are performed with precipitation ; ideas are incompletely developed and follow each other in disorderly array.

Illustrations of the incoherence of chronic maniacs are abundant in Asylum wards, and are not without physiological interest. The female patient, whose portrait is here given, when asked what day it was, replied as follows, her answer being taken down at the time :—' This is the 30th of October or the 1st of November.—What is noted in this month ? Mary Ann left one place and went to another.—What else ? She went to church in a phaeton, but I can make hairs neither black nor white, and that is why she swears—Old dogs never swear, and Mary Ann is three years older than she was. But can I read all the impulse that man puts into that little finger that makes the hand so weak ?—I had not much schooling—That mother of mine went sick, when I was but a slave on the floor with diamonds ; but the hardest diamonds wear out in time and get dirty.—Where are you going to now ? To the post-office—Put roasted potatoes in the bonfire where they burn the logs up in the November gunpowder plot—That mother of mine and father were married in November on a rainy day and went to church in a phaeton, and called at the post-office for a letter—On the 12th of November I scoured the floor of that old school ; the floor and the door-step with sand—Sand is sharp, and so are diamonds—They said they were cocoa-nuts, but I didn't see any—An old door-mat, no doubt—Cabbage is a vegetable, and they left it on the door-mat, and also on the hearth-rug—There was a mouse's nest in the hearth-rug—They were beautiful and honest, and said they wanted a walking commission, and were deaf and couldn't hear—Do I pray over the Bible ? And they raised their hands and played the concertina, and she wore her bonnet in church.'

Perhaps a still better example of the incoherence of

chronic mania is afforded by a letter handed to me by a male patient some time ago :—' I William was born and was stolen for the rights of old England, laws, rules, and regulations, for the Queen of England, for the Queen of Ireland, France, Spain, or Dublin. Cannon-shot and musket-ball, what I call the artillery. Silver spoon or gold china. The drum shall beat and the fife shall play for recruiting or marching away. Milking-cows, doctors of divinity and governors playing " God Save the Queen." Long live our noble King William the Fourth. We sing God save the Gracious Queen with a white cockade in her hat. I was brought up with a silver spoon in my mouth. An eye for an eye and a tooth for a tooth. Rule Britannia, Britannia Rules the Waves, Britons never shall be slaves. But all may be slain in the King's Head Inn, or the Forester's Arms, or the Nag's Head, or in the Grand United Order of Odd Fellows, Bold Robin Hood, the forester so good. All horned-cattle, Little John, Will Scarlett and his man Friday. What do you call tobacco smoking ? Let the bumper toast go round. Here's a health to all good lasses. May they look in their glasses. Not a man in this world has a right to please himself, without being watched as a cat watches a mouse. Pluck at an apple instead of a crab, because they are so much sweeter. Cheap land and cheap silver, and never despise a man who wears a ragged coat. Deeply wailing, still the tree of love I see. The brightest and the best is the morning star. The sun was made to rule the day, and the moon and stars to govern the night, and the darkness comprehended it not. When I was a little school-boy we used to get rewards every Sunday. They are so enticing for scholars. Servants be obedient to your masters. Amen.'

The excitement of certain areas of the brain causes a large development of nervous energy, which expends itself in producing an equivalent amount of new thoughts and emotions in unusual arrangements, and which overflows also in motor currents discharged through various classes of muscles, and notably through the vocal apparatus. Thus chronic maniacs are in a state of perpetual mental and bodily restlessness. They talk to themselves, they chatter throughout

the night, they dance, clap their hands, gesticulate, indulge in shouts of laughter, or tear their clothing to pieces. The length of time during which they can continue to discharge nervous energy uninterruptedly in this excessive, and almost semi-convulsive manner, is truly astonishing. I have had under my care a chronic maniac who worked energetically as a navvy all day, and who sat up in bed talking and shouting all night, for six months together. During these six months he was never known to sleep. By day he manifested not the slightest drowsiness, and by night he was never caught nodding. The night attendants visited him hourly, and at every visit he was in the same position, propped up in his bed, and at the same occupation, pouring out vociferously a torrent of incoherence.

Not less remarkable than the length of time during which unintermitting excitement can be kept up in chronic mania, is the smallness of the effect which the long-sustained and severe exertion and the protracted sleeplessness exert upon the bodily health. Exhaustion is rare in chronic mania. The man to whom I have alluded had not lost weight after his six months' noisy vigil, and many patients who are similarly watchful and turbulent may be seen to be well nourished and in good health, the influence of chronic mania in this respect being very different from that of acute or recurrent states of morbid mental exaltation. Nor does the brain seem to suffer much from the strain imposed upon it. When life is cut short during the course of chronic excitement by any intercurrent disease, that organ is seen to be of almost normal appearance. Whatever changes in it may be revealed by microscopic investigation, none are perceptible to the unaided eye. The membranes are not thickened ; the arachnoid is not opaque, nor is the pia mater adherent to the grey matter beneath. The convolutions are not wasted, nor is their grey matter thin and pale on the one hand, nor swollen and red on the other. No change in consistence can be detected in the medullary substance, nor are the vascular walls perceptibly altered in structure. Of all brains, in short, of chronic lunatics, those of chronic maniacs present the least obvious departures from the normal standard.

In those grave cases of chronic mania that merge into consecutive dementia, impairment of physical health of course often occurs. As the ideas become more and more fragmentary, and as the whole powers of mind are wrecked, bodily decrepitude sets in. But in other cases in which the mania is not of the severest type, and in which fatuity only supervenes very gradually, little or no somatic deterioration can be noticed. The two patients whose portraits accompany this paper have been in a state of chronic mania for five and six years respectively, and they are now in robust health, and show no signs of physical degeneration. Neither do their countenances betray mental degeneracy. Their features do not betoken weakness and vacuity, but they wear that expression which is most common in chronic mania, and which is indeed typical of it—an expression of mingled mirth and mischief. Whether it be that chronic maniacs are tickled by the incongruity and unaccountableness of their own ideas, or that they have a sense of exuberant joyfulness, or that they are merely manifesting the excitement of some cerebral centres of movement, it might be difficult to determine; but certain it is that they laugh inordinately, and that there are generally smiles of some kind on their faces. The corners of the mouth are drawn somewhat backwards and upwards, owing to contraction of the great zygomatic muscles; the upper lip is shortened by contraction of the levatores labii superioris, and a few fine folds are developed round the eyes by slight contraction of the orbicularis palpebrarum, while the bright and sparkling eyes are moved rapidly about or look furtively out from beneath the eyebrows. It is this expression that has been caught in our illustrations, that is habitual in the patients depicted, and that of all expressions is most characteristic of chronic mania, especially when it is associated with negligence in dress, or with peculiar attitudes.

But of course there is no one expression that is constant in this ever-changing malady. Grief, rage, terror, scorn may be signalised by the features in rapid alternation, but the expression described is undoubtedly that which is most frequent and characteristic. To that expression there is not

infrequently added an element of surprise or astonishment, which is not, however, seen in our illustrations, although the male patient from time to time exhibits it. That expression is produced by marked prominence of the eyes, due probably to vascular fulness, and by raising of the eyebrows, owing to contraction of the occipito frontalis muscle. In some chronic maniacs there are habitual rapid movements about the eyes, such as blinking or sudden elevation of the eyebrows.

As has been already said, chronic maniacs are invariably restless, and indulge in numerous unmeaning and curious movements. Some of them attitudinise, others deal destruction to the clothing and furniture around them, others again are perpetually running from place to place. Some of them collect useless articles, such as pebbles and leaves ; and others again bedizen themselves with any cast-off finery that they can lay their hands on.

An excellent picture of chronic mania is to be found in ' Madge Wildfire,' the first conception of whose character, though afterwards greatly altered, was taken by Sir Walter Scott from a person calling herself Feckless Fanny (weak or feeble Fanny), who travelled about the country accompanied by a small flock of sheep. The precision with which the outlines of the portraiture of Madge Wildfire are drawn, and the truthfulness of much of its lurid colouring, mark it as perhaps the best of many admirable studies of madness that are contained in the Waverley novels. The author of these novels, although himself endowed with one of the very healthiest of minds, and though delighting most in grace and picturesqueness, blended with harmonious composure, was yet capable, in his deep strong sympathy with his fellow-men, of some psychologic insight into their most troubled dreams, and of vigorous delineation of their most hideous and distorted freaks and frenzies. ' Your Shakspeare,' says the great critic, ' fashions his characters from the heart outwards ; your Scott fashions them from the skin inwards.' But even allowing that this is so, we are bound to add that Scott by his centripetal method sometimes gets very near the heart, and that he at once touches it, or all that remains of it, when, as in many phases of madness, it is worn on the sleeve. Then in matters

of physiognomy the skin is closely concerned, so that we may expect edification from Scott with reference to the outward bearing and surface-markings of those who are possessed by strong or agitating passions, whether fixed or of 'the borealis race.' He has certainly caught with rare truth the most familiar features and deportment of chronic mania :—

' The officer retired, and introduced upon his return a tall strapping wench of eighteen or twenty, dressed fantastically, in a sort of blue riding-jacket, with tarnished lace, her hair clubbed like that of a man, a Highland bonnet and a bunch of broken feathers, a riding-skirt (or petticoat) of scarlet camlet, embroidered with tarnished flowers. Her features were coarse and masculine, yet at a little distance, by dint of very bright wild-looking eyes, an aquiline nose, and a commanding profile, appeared rather handsome. She flourished the switch she held in her hand, dropped a curtsey as low as a lady at a birth-night introduction, recovered herself seemingly according to Touchstone's directions to Audrey, and opened the conversation without waiting till any questions were asked. . . . . "Ou ay ay ay—but a's forgotten now," replied Madge. " Ye see, I spoke to them myself, and tauld them byganes suld be byganes—her throat's sair, misguggled, and mashackered though; she wears her corpse-sheet drawn well up to hide it, but that canna hinder the bluid seiping through ye ken. I wussed her to wash it in St. Anthony's Well, and that will cleanse, if onything can—but they say bluid never bleaches out o' linen claith—Deacon Sanders' new cleansing draps winna do't—I tried them myself on a bit rag we hae at hame that was mailed wi' the bluid o' a bit skirling wean that was hurt some gait, but out it winna come—Weel, ye'll says that's queer; but I will bring it out to St. Anthony's blessed Well some braw night just like this, and I'll cry up Alie Muschat, and she and I will hae a grand bouking-washing, and bleach our claes in the beams of the bonny Lady Moon, that's far pleasanter to me than the sun;—the sun's ower het, and ken, ye cummers, my brains are het enough already. But the moon and the dew and the night wind, they are just like a caller kail-blade laid on my ·brow, and

whiles I think the moon just shines on purpose to pleasure
me when naebody sees her but mysel."

'This raving discourse she continued with prodigious
volubility, walking on at a great pace. . . . . Madge, who
had looked very pensive when first she stopped, suddenly
burst into a vehement fit of laughter, then paused and
sighed bitterly ; then was seized with a second fit of laugh-
ter, then fixing her eyes on the moon lifted up her voice and
sang. . . .'

In these passages, and in many others which are too long
to quote, are contained accurate representations of traits of
chronic mania.